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# LECTURES ON GIDDINESS

AND ON

# HYSTERIA IN THE MALE

NUNQUAM ALIUD NATURA, ALIUD SAPIENTIA DICIT.

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# HYSTERIA IN THE MALE

BY

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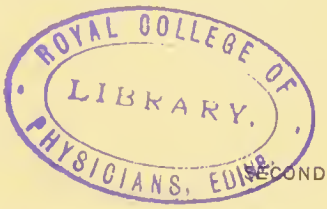


SECOND EDITION

EDINBURGH AND LONDON:
YOUNG J. PENTLAND
1898

EDINBURGH : PRINTED FOR YOUNG J. PENTLAND, 11 TEVIOT PLACE AND
38 WEST SMITHFIELD, LONDON, E.C., BY R. AND R. CLARK, LIMITED.

# PREFACE TO SECOND EDITION

The Lectures on Giddiness have been long out of print, and are now presented with some additions. I have thought it well to publish along with them an account of a case of Hysteria in the Male, exhibiting some features not previously described. It originally appeared in the Edinburgh Hospital Reports for 1894.

T. GRAINGER STEWART.

19 Charlotte Square,
Edinburgh, *July* 1898.

# CONTENTS

## LECTURE I



## LECTURE II



## LECTURE III

## HYSTERIA IN THE MALE

# LECTURES ON GIDDINESS

AND ON

# HYSTERIA IN THE MALE

# ON GIDDINESS

## LECTURE I

Gentlemen,—We have frequently met with giddiness as a prominent symptom in cases which we have studied in the wards, and I promised that I would take an opportunity of discussing the subject in the lecture-room, illustrating my observations by cases which I might be able to show you or to describe. I find it useful, sometimes, to take up a symptom in this way rather than to discuss individual cases or groups of cases, as we usually do in this lecture-room.

### *Description of Giddiness*

Giddiness is a sensation with which we are all familiar; but which it is difficult to describe. Its essential feature is the feeling of uncertainty of our position in space relatively to surrounding objects. Sometimes one feels as if objects were whirling round more or less rapidly; sometimes one seems to be moving in the midst of them; and that apparent movement may be round a vertical or a horizontal axis, to one side or other, forwards or backwards; or there may be a sensation of sinking downwards; or the movement may appear confused or irregular. It may also be slow or rapid.

### *Associated Conditions*

Giddiness is often associated with other disorders of sensation, such as nausea, deafness, tinnitus aurium, or other subjective sounds, dimness or other changes of vision, faintness, palpitation, hallucinations, and is sometimes followed by loss of consciousness. But it is constantly associated with impairment of equilibration. This is readily explained when we consider that it is the result of derangement of a function essentially related to equilibration,—disturbance of equilibration being, so to speak, the motor aspect of the sensation of giddiness. For equilibration we require the co-ordinated action of many groups of muscles. This co-ordination is, like others, guided by sensory impressions, and these impressions are derived from different sources.

This is so important as to deserve re-statement in a slightly different form and with certain additional points. Giddiness is, then, the sensory correlative of staggering: staggering is the motor correlative of giddiness. Very commonly they co-exist, and either may cause the other. Thus in almost every case of giddiness, staggering shows itself more or less distinctly, and in some cases staggering induces giddiness. It is easy to understand the former of these facts—for steadiness in standing and walking require not only motor action but sensory guidance, and if the sensory impression be disturbed the movement is necessarily interfered with. It is more difficult to see why staggering should sometimes cause vertigo, but the explanation seems to lie in the fact that nervous actions habitually associated with one another may either of them awaken the other, and especially when that other is of the nature of a sensation.

*Equilibration an acquired Power dependent upon Sensory Impressions*

Neither the power of equilibration, nor the power of appreciating the impressions by which it is guided, is inborn in man. Each is gradually developed by processes of education. The senses which are trained to subserve it are touch, sight, the muscular sense, probably an articular sense, and a visceral sense, along with the great organ of special sense for equilibration —the semicircular canals. Of some of the impressions we are conscious, of others not. Of sight and touch impressions we habitually take cognisance, or at least we may do so; of the other kinds of impressions we make use without any act of intellectual perception. It is at once apparent how sight subserves the purposes of equilibration, and it becomes especially noticeable when the other sources of sensory impressions are impaired. You have often seen this in cases of locomotor ataxia. You have noticed how helpless the patient becomes when his eyes are closed. The importance of touch, and particularly plantar touch, is equally familiar to you, being often illustrated in the same class of cases. The impairment of the muscular sense, I have sometimes shown you, is at least as important as those already mentioned. The articular and the visceral are less obvious, but I think are real. I shall not dwell upon them, but the special sense for equilibration, which has its seat in the semicircular canals, requires fuller notice.

Let me remind you of some anatomical facts and physiological explanations. The canals are situated in the petrous portion of the temporal bone. They are three in number, and communicate by means of five

openings with the vestibule. Each canal has a dilated or ampullary end, and each by its ampullary end opens into the vestibule, while two of them have their non-ampullary ends united, and communicate with the vestibule by a common opening. These canals occupy different planes, and are known as superior, posterior, and horizontal, according to their position. Within the bony labyrinth there is a membranous labyrinth, the membranes being much smaller than the canal, except at the ampullary ends. Between the bony canal and the membranes is the perilymph, with connective tissue, blood vessels, and nerves. Within the membranous canal is the endolymph. A branch of the portio mollis of the seventh nerve is distributed to a crescent-shaped ridge near the middle of each ampulla. The terminal nerve structures are hair-like cells, which project into the endolymph, and which must obviously be affected by changes in it. Through the chain of bones the labyrinth is connected with the tympanic membrane. The handle of the malleus is fixed into the tympanic membrane, while its head articulates with the incus. The latter, again, is connected with the stapes as well as with the posterior wall of the tympanum. The stapes is fixed into the fenestra ovalis, and by its movements produces alterations in the state of the fluids in the whole labyrinth.

In 1828 Flourens discovered that the semicircular canals have something to do with the maintenance of the equilibrium of the body. He found by experiment that section of a membranous canal was always followed by movements of the head, or even of the body, rotating about an axis at right angles to the plane of the divided canal. On section of the horizontal canals, the animal moved its head rapidly from side to side, and tended to spin round on a vertical axis. When the posterior

canals were cut, rapid movements backwards and forwards took place, and the animal showed a proclivity to turn somersaults, from before backwards. When the superior vertical canals were cut, similar movements occurred; but the tendency now was to turn somersaults from behind forwards. Section of all the canals induced peculiar irregular movements. The movements always became worse if the animal was excited or disturbed. If one side only was cut, the abnormalities gradually subsided; if the canals of both sides were injured, the derangement of motion was permanent. These were clearly not the mere results of irritation, seeing that the symptoms continued long after the external wounds had been healed.

A great advance was made by Goltz when he suggested, in 1870, that pressure of the endolymph gives rise to sensory impressions, and that, according to the position of the body, the pressure varies by gravitation in different portions of the series of canals. He showed that the sensory impressions bear an important relation to equilibration, and that by altering these impressions he could produce results similar to those which follow section of the canals. He fixed the heads of pigeons in peculiar positions, tying, for example, the head down upon the breast, and found that equilibration was thereby impaired.

The next advance was made by Cyon in 1872, who repeated and confirmed the former observations, and formulated the theory that from the semicircular canals we derive impressions as to the position of our heads in space, each canal being definitely related to one of the dimensions of space, that these impressions guide us in equilibration, and that failure of equilibration may be due to disorder of their functions.

In 1875 Professor Crum Brown published an

interesting account of some experiments which he had made upon the subject. He pointed out that if the body be passively rotated, one can, with the eyes bandaged, determine pretty accurately the amount of movement made; that if such passive movements be continued beyond a certain time, the feeling is lost, but that if the rotation be suddenly arrested, the sensation of rotation continues. He suggested the theory that the sensations are due to alterations of pressure in the endolymph, not from mere gravitation, as had been supposed, but from the formation of currents (or, more strictly speaking, the production of a strain) in the endolymph, that fluid being conceived to lag behind in the rotational movement of the head. He pointed out how well such a theory suffices to explain the facts discovered by himself, as well as those previously known. Moreover, he remarked, that as each canal has but one ampulla, and as the same organ of sense can produce sensations differing in quantity only, and not in quality, it is probable that one canal will respond only to the rotation round one axis, and in one direction, because the sensation of rotation in the opposite direction is qualitatively different. Hence he concluded that the semicircular canals are paired organs, and that each canal corresponds only to rotation in a single direction. The same theory was promulgated independently, and at about the same time, by Professors Mach of Prague and Breuer of Vienna.

Some later experiments of Cyon throw doubt upon this kinetic theory, but cannot be held, in my opinion, to have disproved its principle.

You perceive, then, that we have abundant proof that the semicircular canals are sensory organs subserving equilibration, and that we are gradually acquiring definite ideas as to how their functions are performed.

Besides these peripheral structures which guide us in our equilibration, there are, of course, conducting fibres, and nerve centres which subserve the functions. Changes in any part of these structures may suffice to produce the sensory and the motor abnormality. We shall first consider the peripheral causes of giddiness, and then those due to changes in the conducting fibres and nerve centres.

In normal equilibration we guide ourselves by information derived from all the peripheral sources to which I have referred—and in normal conditions they all harmonise—the information derived from sight, from touch, from the muscular sense, from the labyrinth, all correspond. When they do not, giddiness results. Observe, then, that peripheral giddiness is usually due to

*Contradictoriness of Sensory Impressions*

Sometimes the contradictoriness is due to external conditions. This is especially true regarding sight. If you visit the famous Leaning Tower of Pisa, you will most likely experience, as you ascend and descend its stair, a peculiar, uneasy feeling of slight giddiness. On experiencing this sensation, it occurred to me that it was probably due to contradictoriness of impressions derived from sight, and from the other sources of impression guiding equilibration; and I found that it was so, for, by slightly altering my attitude, the feeling at once disappeared. In the tower there were three others along with me, all of whom felt the same uneasiness, and all were freed from it by altering their posture very slightly. I am satisfied from what I saw when there, and from what I have heard since, that that peculiar giddiness is felt by very many people. Some of you may have visited, and many of you will hereafter visit, the famous

tower. When you do so, you will be able to verify what I have told you.

### *Cliff or Tower Giddiness*

But you have all had opportunity of observing another variety of the feeling—what we may term cliff or tower giddiness. A large proportion of people suffer more or less from an uneasy sensation of giddiness whenever they stand upon a tower or a cliff, or even a steep stair or a hillside. You remember the description of it in *King Lear:*

> How fearful
> And dizzy 'tis, to cast one's eyes so low!
> . . . .
> I'll look no more;
> Lest my brain turn, and the deficient sight
> Topple down headlong.

It is due, I take it, to contradictoriness of impressions. They have been accustomed, under ordinary conditions, to receive simultaneously impressions from the plantar surfaces, from the muscular, articular, visceral, and labyrinthine structures, with visual impressions of solid ground before them; but when standing on a tower or cliff they find the visual impressions contradicting the others, and so the peculiar feeling arises. The liability to this kind of giddiness varies in different individuals, and is determined by the sensitiveness of the nervous system, on the one hand, and by the degree in which the individual has been accustomed to trust to visual impressions for equilibration, on the other. You will find the feeling aggravated if you look downwards to where, according to experience, the solid ground should be, and absent or diminished if you look in other directions. Some people are so sensitive in this respect

that they cannot with comfort sit in a gallery of a church or theatre.

It is well worthy of notice that a psychical or mental element mingles with the merely sensory to a greater or less extent. I have known a person not liable to cliff giddiness begin to suffer from it when a group of young people who accompanied him on a hill expedition came, as it seemed to him, dangerously near the edge of a precipice. On the other hand, the liability to the symptom varies with the state of the general health, and in particular with even functional disturbance of the stomach and the liver.

### *Agoraphobia*

A very curious condition, manifestly allied to this, is known as "Agoraphobia." Benedikt described it in 1870; Westphal in 1872. Its essential feature is, that while the subjects of it can walk quite well in a narrow lane, or even in an ordinary street, they become uneasy, and experience either giddiness or a peculiar feeling allied to it, when they attempt to traverse a wide market place or square, such as narrow lanes often open into in continental towns.

Let me describe by way of illustration one of Westphal's cases. The patient was a commercial traveller, aged thirty-two, of middle height, slender, healthy looking and mobile, and complained that he suffered greatly when he tried to walk across open squares; when he attempted to do so, a peculiar uneasiness, situated mostly in the head, but attended by violent action of the heart, was induced. On attempting to cross a square he felt as if it were miles wide, and often he trembled all over; the more he approached to the houses at the side the less was his uneasiness; a stick was no

help to him, but walking arm in arm in lively conversation helped him considerably. If absorbed in thought, he could sometimes accomplish the feat; if he kept close to a carriage crossing the square he could manage pretty well; he had to deny himself the pleasures of the Thiergarten, and could not by any effort make his way to Charlottenburg (a public garden and favourite promenade in the neighbourhood of Berlin). The same uneasiness was felt in some measure if he had to walk beside a blank wall or when the shops were shut; he usually supped in an eating-house, and had to help himself home by ingenious devices; he waited till he saw people going in his direction, and followed them up, or he followed closely behind a conveyance. When asked what he would do if left without any of these external guides, he said that the very thought appalled him, but that he would probably throw himself on the ground and hold on by the grass. In his frequent journeys he had constantly to avail himself of stratagems of war, so as to be prepared under any circumstances to get assistance. On entering theatres and churches a similar uneasiness occurred at one time; but that difficulty disappeared. He could sail without much discomfort in a large vessel; not in a small boat; and could only walk at the seaside in places where the houses come close to the shore. He lived in constant dread of these uneasy feelings. He described them as differing from giddiness and from the feeling experienced when looking from a tower. He benefited by moderate use of alcoholic stimulants. The disease began in his twenty-seventh year. The nervous system was not otherwise abnormal; but there was a slight want of symmetry of the two halves of the body. There was no marked history of nervous disease in his family.

In this patient it will be observed that there was

abundant evidence of the peculiarity of the nervous organisation, but the point to which I wish to draw your attention is the inability to walk across an open square, and the peculiar sensation experienced by the patient. Benedikt speaks of the sensation as giddiness, and although Westphal has drawn a distinction, as this patient did, between ordinary giddiness and this special uneasiness, I think we are warranted in believing that the feelings are closely allied.

Various explanations of the condition have been attempted, but none of them seems to be quite satisfactory. A reasonable explanation of one element of the trouble lies, I think, in the supposition that these patients, with the nervous system morbidly sensitive, have from some peculiarity acquired the habit of guiding themselves in their equilibration by reference to near vertical lines; and that, when these vertical lines are awanting, they feel as others do when standing on the edge of a cliff. Notice the fact about the methods they devise for getting across squares. If they can get behind some vehicle, they can walk well enough; the vertical line of the vehicle suffices to guide them.

I have met with one case of another condition allied to these. I know a lady who experiences great discomfort if she sits in a building with a lofty ceiling. She always requires to sit under a gallery in church; and the explanation is, that to her it is essential that she should have the impression of a plane not far off above her head. In these forms of giddiness you observe that, on the one hand, we have certain nervous susceptibilities more or less pronounced, combining with certain external conditions to induce this sensation.

I shall now refer to some causes of giddiness with which you are probably all more or less familiar, as they

occur in a large proportion of people when placed under certain conditions.

### *Giddiness of rapid Rotation*

There is, first, the giddiness resulting from rapid rotation,—the waltz giddiness. This appears to be due, not so much to the contradictoriness of impressions, as to powerful impressions made upon the special centre for equilibration, the rotation producing changes in the semicircular canals, well explained, as we have seen, by the kinetic theory of their function. Every one naturally becomes giddy with rapid rotation, and with this, under ordinary conditions, equilibration fails. But one may be trained to equilibrate, notwithstanding the labyrinthine impression. All physical education consists in learning to co-ordinate muscular movements. In learning to waltz the child has not only to learn to co-ordinate certain groups of muscles, but has to learn to do so while he is receiving misleading impressions from sensory organs on which he is accustomed to rely. He has to guide his movements by the plantar, muscular, articular, and other impressions, and to time them by the music, disregarding his labyrinthine impressions altogether.

### *Swing Giddiness*

Swing giddiness is not unfrequently experienced by children when they are enjoying that exhilarating amusement. The giddiness you will find, if you analyse it, is due to a variety of causes. There is obviously the constant changing of the visual impressions. But this is not all. The feeling is most marked when the swinger is descending. Now, during the

descent, a very peculiar visceral sensation is experienced. It appears as if the body, as a whole, descended at a greater rate than the abdominal viscera, and as if they followed; and as they followed, a certain friction took place between the peritoneal surfaces—the visceral sensation being attended by a quite peculiar uneasiness. It is probable that, in the cranium and in the semi-circular canals a similar action may take place; but these we cannot so readily determine as the visual and the visceral impressions.

### *Sea-sickness Giddiness*

In sea-sickness, with its attendant giddiness, we find many elements contributing to the result. There are visual impressions—some very susceptible persons becoming affected even when they look at the sea; but every one who suffers from it feels his discomfort increased if he watches the varying elevations of the bulwarks of the vessel and the surrounding waves, and feels the better of looking another way, or of closing his eyes. But in this condition also the visceral impressions are most important. As the vessel sinks into the trough of the wave the body seems to descend as in the swing, the viscera appearing to sink downwards at a slower rate than the body generally. And here, as on the swing, this is attended by a terrible uneasiness. I suppose that one of the great advantages of the sea-sick invalid laying himself down at full length is that he thereby diminishes the visceral movement. With the labyrinthine impressions also the nerve centres must be sorely puzzled, for from moment to moment the impression varies as the vessel rises or sinks, or rolls from side to side. The muscular sense, also, is brought into most unusual conditions, as is also the articular sense. But

one other source of sensory impressions is also at fault, for the plantar pressure varies from moment to moment. At one moment, as the vessel sinks into the wave hollow, it seems as if the vessel were falling from beneath the feet, and there is almost no plantar impression; and then, again, as the vessel rises, the pressure becomes greater. In such conditions it is clear that the sensorium receives a great variety of ever-varying and contradictory impressions.

### *Giddiness from Ocular Causes*

But giddiness may be due to abnormalities of the sensory structures themselves. Notice, first, some ocular causes. In illustration of this I shall recount to you a typical case seen by some of your predecessors. When I first took charge of the clinical wards as professor, I found a patient who was unable to walk or even to stand on account of fits of giddiness, with which he was frequently seized. On examining him I found no evidence of central nervous disease, but observed that when he took his giddy turns there was always a rapid oscillating movement of his eyes. It occurred to me that oscillation or nystagmus might cause the giddiness, and I held his eyes fixed by slight pressure. This cured the giddiness in a moment, and while it was kept up the giddiness never recurred. On further inquiry I found that the patient was a miner, and that his case was a typical one of what has been described as miner's nystagmus. The patient continued under observation for some time, but no good was done except by the mechanical treatment.

The giddiness in that case was manifestly not due to any central disease, but was simply a result of the contradictory impressions resulting from the movement

of the eyes. The disease is now pretty well known. It is due to spasm from long-continued muscular strain —is indeed a form of trade spasm. The miner while at work lies on his side, and opens up the coal-seam above and beyond his head. He works also in a dim light. He has, therefore, to strain his eyes greatly, and to keep them in an unnatural position, and this nystagmus, with its attendant giddiness, occasionally follows.

You may ask me whether nystagmus always induces giddiness. Certainly it does not. It sometimes is merely associated with giddiness, the two depending upon a common cause, as in cerebellar disease. Sometimes, again, it results from cerebral disease, sometimes from disease of the retina, sometimes from disease of the cornea, occurring in childhood. In such cases, giddiness does not result, because the sight is so much impaired that the patient learns not to be dependent on the visual impressions for guidance. This is especially the case when the cause comes into operation in early life.

Paralysis of any of the external muscles of the eye, when of recent origin, frequently leads to giddiness from double vision. You will often see this fact illustrated in the eye wards, but I may mention an example of it which occurred in the case of a patient whose illness I shall describe to you later on in connection with intracranial lesions. The patient, a young man of nineteen, naturally of robust constitution and a rather eminent university athlete, had sustained an injury to the head, and soon afterwards observed that he saw double, and had a constant feeling of giddiness. He next discovered that if he closed one eye, his giddiness disappeared. It was manifest, then, that the giddiness resulted from the contradictory ocular impression due to squint, which again in its turn was due to paralysis of one of the

ocular nerves. The test which I should advise you to apply in such a case is the simple one of getting the patient to close one eye; if this cures the giddiness you may be quite sure that it is of the nature we are now considering. Paralysis of other muscles of the eye may, of course, lead to the same results.

Any one of you may experience for himself the sensations of ocular giddiness by making the following simple experiment. With the finger press one eyeball inwards towards the nose so as to cause diplopia. Then keeping both eyes open attempt to walk along a straight line when you will find it absolutely impossible to do so, owing to the giddiness which, with its attendant staggering gait, at once results.

Sometimes eye giddiness occurs without there being any perceptible squint. I know a literary man, an omnivorous and very rapid reader, who is taken with giddiness and frontal headache whenever he looks upwards, as for example to take books from a high shelf of a library. It is the more marked if he stands on the top of steps, and that although he is extraordinarily free from any tendency to cliff giddiness and never in his life experienced the slightest vertigo from rapid rotation as in waltzing. The effort of using the eyes somewhat persistently turned up in an unusual way induces so marked an attack that he is obliged almost at once to desist.

I must defer till next lecture an account of the most important of all the peripheral forms of giddiness, that due to alterations in the semicircular canals—Menière's Disease.

## LECTURE II

GENTLEMEN,—Our last clinical lecture was devoted to the subject of giddiness, and after describing to you the nature of the sensation, I discussed certain of its causes, —viz., those due to altered conditions outside the body, and those which result from abnormal peripheral impressions. At the end of the hour I was about to explain the relationship of giddiness to diseases of the semicircular canals.

### *Labyrinthine Vertigo*

Of all the forms of giddiness from peripheral causes, the most interesting and important is that group due to causes situated in the semicircular canals, which are commonly known as Menière's Disease, or Labyrinthine Vertigo. Dr. Menière, as superintendent of the Institution for Deaf and Dumb in Paris, enjoyed exceptional opportunities of studying such conditions, and by the light of the discoveries of Flourens in regard to the semicircular canals, he concluded that many attacks which had been considered cerebral, were in reality due to disease of these organs. In 1861 he brought his observations before the Academy of Medicine in Paris.

As a type of this condition, I will cite to you one of his most striking cases. A young woman travelled on a winter night on the outside of a stage-coach, and was exposed to great cold. She was at the time menstruating.

She suddenly became completely deaf. When received into the hospital wards, the principal symptoms were constant vertigo and vomiting induced by the slightest effort to move. She died on the fifth day. At the autopsy it was found that the brain, cerebellum, and cord were free from morbid change. As the patient had all at once become deaf, although she had her hearing prefect up to the time of the attack, Menière removed the temporal bones, so that he might be able to ascertain the cause of the deafness. The semicircular canals were the only part of the labyrinth that presented any change; they contained a reddish-coloured plastic lymph instead of the fluid of Cotunnius. From such observations Dr. Menière drew his first description of the disease.

That it is not always fatal the first case which came under my care may serve to show. An apprentice boy was brought into my wards suffering from most of the symptoms of Menière's disease, namely vertigo, faintness, vomiting, and reeling gait, and roaring or humming noises in the ears, with deafness of one side. On inquiry, I found that the illness had come on suddenly, and been caused by a violent blow on the side of the head which the boy had received when at work. In a few days improvement set in, and in a short time the patient was well. I supposed that, in consequence of the blow, some slight hæmorrhage had taken place into the semicircular canals, and that the blood so effused was speedily absorbed. Whether this view of the case be correct or not, you will remember that a very speedy subsidence of the symptoms may occur. In some cases, while life is not endangered, the patient continues to suffer for long periods—even for many years—and life is made almost intolerable by the discomfort it occasions.

Some recent writers on the subject, such as Dr. M'Bride, recognise several varieties of Menière's disease.

And among these I mention, first, the forms due to *changes in the external ear*. Some people experience all the symptoms of Menière's disease if they have their ears syringed, and often accumulations of wax, or of eczematous secretions, produce the same result, if they be so situated as to press with force on the tympanic membrane.

I show you to-day a patient who has several times consulted me of late. She is fifty-six years of age, and has come complaining of giddiness, pain in the head, with dulness of hearing, a feeling of oppression within the cranium, and a singing or buzzing sound in the ears. Her hereditary and social history are unimportant; but she has lately passed through very severe trials, to which she ascribes her illness. She has two sets of abnormal sensations, one constant, the other occasional or paroxysmal. The constant feeling consists in noise in the ears, uneasiness in the head, deafness, and general discomfort. The paroxysmal consists in severe fits of giddiness, with palpitation, faintness, and coldness. The giddiness is peculiar, in respect that she feels an inclination to fall backwards; and a friend who accompanied her to the Infirmary says that this is not a mere sensation, but that she actually tends to fall back. If she is able to sit down and press the back of her head against anything, the giddiness is at once relieved. Her ears were carefully examined by Dr. Graham Brown, who found that the Eustachian tubes were not readily permeable, while there was a large mass of wax in the right meatus. On this being removed, the buzzing at once ceased; and during the week that has elapsed now since this treatment was adopted, she has felt almost well, and has certainly had none of her paroxysmal attacks. Here, then, is an example of a group of formidable symptoms depending mainly upon a cause situated in the external meatus.

The second group is that *due to disease of the middle ear*, which is generally considered to be the commonest cause of the symptoms. As an illustration I shall cite the case of a patient who was under my care in the Infirmary some years ago, and in which I had the benefit of Dr. M'Bride's advice and assistance. The following account is in the words of that gentleman:—

"W. H., *æt.* fifty-eight, complained of giddiness and staggering. He is a blacksmith by trade, and exposed to sudden changes of temperature; and to this he attributes, in a great measure, his illness. For five years his hearing has been getting gradually worse. Before the patient applied for advice, he had two very severe paroxysms of vertigo, with an interval of two days between them. What occurred on these two occasions seems to have been that the patient felt intensely giddy, heard noises in his head, and lost consciousness. This happened for the first time on a public thoroughfare, and when he recovered his senses he found himself seated and being attended to by bystanders. He then felt sick, and everything round him seemed to turn (direction not observed). He staggered home, and when he got there vomited and fell asleep. On awaking he felt better, and next day went to work, feeling well. On the day following, however, he had a repetition of the attack. Since then the patient has been troubled with constant giddiness and staggering towards the left. A mixture of iodide and bromide of potassium seemed to have little or no effect on the symptoms. Except for the giddiness and staggering, the patient seemed in good health.

"The examination of the ears gave the following results:—Watch (normally heard at thirty inches) not heard in contact with either auricle. Conversation is, however, fairly well heard, and the tuning-fork is well

heard in both ears by bone conduction. Tympanic Membranes—Right membrane opaque, with vascular injection along the handle of the malleus. Anteriorly and superiorly there is a dark atrophic patch. Left membrane indrawn, with well-marked folds. Politzer's method only succeeded in forcing air into the left tympanum. After this, inspection showed the corresponding membrane flattened out, the folds having disappeared. Air could only be driven into the right middle ear through the Eustachian catheter, and auscultation during the process revealed a distant gurgle, indicating probably an accumulation of mucus in the pharyngeal orifice of the right Eustachian tube.

"On the first attempt to inflate the tympana by Politzer's method, a very extraordinary effect was produced. As stated, air only entered the left middle ear; but whereas, before the operation, the patient had great difficulty in standing and walking, even with assistance, after the inflation he declared himself greatly relieved, and was able to walk about alone and unsupported. The subsequent treatment consisted in astringent applications to the throat and nasopharynx, which were in a catarrhal state, inflation of iodised steam into the tympana, and occasional use of the air-bag and Eustachian catheter. The result of this treatment was that in a week or two the only symptom of vertigo left was that looking down suddenly produced slight giddiness. The hearing also improved considerably; that is, he could hear a watch, which was previously altogether inaudible, on the right side in contact with the auricle, and on the left just off the ear. The tinnitus also, from which he suffered, was much relieved."

Commenting upon these facts, Dr. M'Bride says: "Now, this case is perfectly typical of what is often known as Menière's disease in an aggravated form. The

vertigo, tinnitus, vomiting, and loss of consciousness, were all well marked. The deafness, however, was most distinctly traceable to chronic middle ear catarrh, pure and simple. There was no organic mischief in the cochlea, as shown by the tuning-fork test, nor in the semicircular canals, as proved by the success of local treatment directed to the middle ear. Moreover, we could trace the nervous phenomena to deficient ventilation of the left tympanum; for whenever air entered it there was an immediate and marked improvement. It was on that side, too, that there was most evidence of altered intratympanal tension in the appearance of the drumhead; and we have before seen that changes of tension in the tympanum produce a more or less corresponding alteration in the endolymph. The case is important as one showing the most marked and aggravated characteristics of Menière's disease, depending upon a curable condition of the middle ear—curable, be it observed, not by empirical inunction, of iodine and application of blisters, but by rational treatment directed to recognised pathological conditions. I have seen a considerable number of cases where staggering and giddiness depend upon middle ear lesions of various kinds, but none so severe as in this case."

In Menière's first case the symptoms were *due to lesions of the internal ear*. The disorder may supervene in various ways quite suddenly, as in the case of Menière, or in that of the apprentice boy, already cited; or very gradually occurring in connection with chronic disease. The condition may also be fatal, or it may pass away quickly, or it may last for a long time without endangering life.

With regard to the treatment of Menière's disease, you must seek to determine the particular form of the malady with which you have to deal. If the cause be

situated in the external meatus, try to remove it. If it be due to obstruction of the Eustachian tubes, seek to have them opened up. If it be due to changes in the labyrinth itself, much less can be done; but by means of counter-irritants and absorbents good results may be obtained. Charcot has spoken strongly of the advantage following the use of quinine in this disease, and remarks that it often seems to relieve the patient from the humming or ringing sounds which are so frequently complained of. Pilocarpine has been found useful in some cases.

### *Giddiness with Disease of Cord*

Lesions of the spinal cord are not very often attended by giddiness, but in cases of locomotor ataxia, it is sometimes much complained of, and I have found it more prevalent in earlier stages of that disease than in the later. It may arise in various ways. Let me describe to you some cases. A very intelligent schoolmaster, resident in a country district in Cumberland, consulted me on account of spinal troubles. I found that he had for several years had slight symptoms of locomotor ataxia; but that in consequence of overwork preparatory to inspection of his school, a degree of actual paralysis had supervened. This paralysis happily passed off in a few weeks under treatment, but the ataxic symptoms persisted. He told me that he very frequently suffered from vertigo, but, on inquiry, it proved to have been present only when he also had double vision or actual squinting. I show you to-day also a patient suffering from locomotor ataxia. You observe that the gait is distinctly characteristic, and that, when his eyes are closed, he tends to fall. He complains much of giddiness, but this giddiness is cured when he closes one eye. It is a result of the double

vision. In these cases then the cause is manifestly ocular, and referable to one of the groups already discussed. But that is not the only cause of giddiness in locomotor ataxia. It seems to result sometimes from changes in the sensory impressions, from other sources, and sometimes perhaps from changes in the brain.

Patients suffering from multiple cerebro-spinal sclerosis often complain of giddiness. It may possibly result from the nystagmus, but certainly cannot be referred in all cases to this cause, for it may occur without nystagmus, and be awanting when nystagmus is present. It is apt to occur in short isolated attacks, and apart from any circumstance which seems obviously fitted to explain it.

### *Giddiness with Intracranial Lesion*

Giddiness arises from a great variety of intracranial conditions, conditions varying both in respect of seat and of lesion. It occurs from lesions of the cerebellum, the crura cerebelli, the pons, the cerebral peduncles, the corpora quadrigemina, and other parts of the cerebrum, and it arises from anæmia, congestion, hæmorrhages, softenings, and new formations. At the same time it must be admitted that cases may be found on record of lesion in any one of the above-named intracranial structures in which no giddiness occurred, and it is equally true that each of the lesions may be unattended by the symptom.

#### (*a*) *Cerebellum*

Sometimes the giddiness appears to be a direct symptom; sometimes it merely results from paralysis, especially paralysis of some of the ocular muscles,

attended by double vision. It is certainly most constantly associated with cerebellar disease. The peculiar swaying or staggering gait, like that of slight intoxication, has long been recognised as a feature of cerebellar maladies, and although the staggering is not always, it is very often accompanied by a feeling of giddiness. It is said to be generally increased when the patient opens his eyes, and relieved when he lays hold of anything. It may be present only occasionally, or when the patient is in the upright posture, but sometimes it is present even while he is lying down. As a good typical case of cerebellar disease, I select one which is recorded by Professor Nothnagel as having been observed by himself. W. P., a servant girl, thirty-six years of age, had complained since the beginning of August 1876 of a frequently recurring feeling of giddiness. It used to come on suddenly, especially when she was walking, or when she rose from her chair, and usually disappeared in a few seconds or minutes. At the same time she used to sway to both sides, but never fell to the ground. From the beginning of September she had severe pain in the back of the head; from the middle of September she had frequently malaise and vomiting. Later on, she had frequently humming in both ears, and she observed an unpleasant saline taste in the mouth and increased thirst. In the beginning of October Nothnagel examined her, and found that intelligence was normal, that there was almost constant occipital pain, that she always lay on her back, because when she sat up she had giddiness and vomiting. The movements of arms and hands were free and strong. The finer movements were easily performed, even when the eyes were closed. There was no abnormality of the muscles of the face or the tongue. All movements of the limbs were rapid and precise, even with the eyes

closed, and the patient knew the position of her limbs, and could point to them. She could walk if she got the slightest hold of anything; but if she were left alone, marked swaying occurred (in no particular direction), with giddiness, choking, vomiting, and the patient tumbled down if not held up. It made no difference whether the eyes were closed or open; without help she could not walk, and even with support she walked unsteadily. When giddy, the patient felt as if objects moved round her. The special senses were normal: the ophthalmoscope showed in the right eye slight venous congestion; in the left, a normal fundus. During October and November her condition remained unchanged; from the end of December she frequently had epileptiform convulsions and double vision. She died on 18th January 1877, within six months of the commencement of the illness. On the post-mortem examination the cerebrum was found to be normal. The cerebellum was found to contain several tumours, involving both sides and also the central part. The crura cerebelli and the corpora quadrigemina were unaffected. In this case, you observe that giddiness was very pronounced, and that it preceded the occurrence of the other symptoms, was not due to squinting, and most likely resulted from the cerebellar disease.

As an instance of cerebellar apoplexy, I shall mention one observed by Dr. Brodribb of Hastings, and briefly recorded in Sir Samuel Wilks' work on the *Diseases of the Nervous System.* A lady, aged fifty-three, had travelled from London to visit a sick relative, and being depressed in spirits had recourse, more than once, to her brandy bottle. After her arrival at the house, she went out for a drive, and on her return she could scarcely walk or stand. She expressed a fear that she was intoxicated, having partaken too largely of brandy, and

her friends were at first willing to coincide in this opinion. After a few hours, however, she fell into a state of coma, and died. The post-mortem examination showed a clot of blood of the size of a pigeon's egg in the right lobe of the cerebellum. Dr. Brodribb, on further inquiry, could not learn that there had been any special loss of motion, or any disturbance of the senses, but merely a want of regulating movement, such as is seen in an intoxicated person.

With regard to the cerebellar reeling, you must observe that it is not always associated with giddiness. I lately had the opportunity of studying closely a case which I believe to be certainly cerebellar. A student, nineteen years of age, and who had been remarkably strong, and, as I was told, a leading athlete in his university, sustained an injury to his head about four years ago. At first this injury was followed by no symptoms, but soon there occurred squinting, with double vision and giddiness. This giddiness, he found, as I have already told you, that he could cure by closing one eye, and it disappeared permanently as the squint passed away, in the course of three or four months. From this time he remained free from any unhealthy symptoms, able to pursue his studies and his athletics, until about six months before he came under my notice. He then observed that in walking he occasionally reeled to one side; and people remarked upon the peculiar gait, ascribing it to intoxication. But, while he thus staggered, he felt no giddiness. He noticed that the staggering was more toward the right side, and that his head tended to go in that direction, and forwards; and he felt just as if he were making the extraordinary movement in order to maintain his equilibrium. He became unable to ride because of the tendency of his head to fall forward, a tendency which he could better overcome when on foot

than when on horseback. Four months before I saw him, although other people constantly remarked upon his peculiar gait, he was able to go about pretty well; indeed, he walked a distance of six miles in deep snow, and appeared to be little the worse of it, so that his muscular vigour was evidently then little impaired. But for a marked tendency to constipation, I found all his organs and functions healthy, except the nervous system. He complained of a feeling of numbness in the limbs after exertion, but sensibility was unimpaired; his sight was good, and the fundus of the eyes normal. But he said that after walking or other fatigue his sight became indistinct. Hearing, taste, and smell were natural. With regard to his muscular sense it was difficult to speak; there was no definite proof of it being impaired, unless it be a proof that he staggered more when his eyes were closed. His skin reflexes were natural, the patellar tendon reflex slightly exaggerated; the organic, not interfered with. His voluntary movements were imperfect, in consequence of a tendency to lurch to the right side and his head to pitch forward. He grasped firmly with the hand, but performed finer movements very imperfectly; in particular, could not write well, especially when tired, or when he had been attempting to study. I think that it can scarcely be doubted that in this patient cerebellar disease has occurred, probably as a consequence of the injury sustained four years ago. The gait is quite characteristic, and differs markedly from that of locomotor ataxia. I satisfied myself that he felt no giddiness even when the staggering was pronounced.

Two interesting cases are recorded by Dr. Fraser of Paisley, referable to the group which has been recently defined by Marie, Sanger Brown, and others as hereditary cerebellar ataxia. In one, which proved fatal, the brain

and cord were carefully examined, and it was found that the cortical gray substance of the cerebellum was little more than half its normal thickness, and that there were gaps at the surface, probably caused by collections of fluid in the membranes; that the white substance was less reduced in bulk, indeed appeared excessive in proportion to the gray; and that the cells of Purkinjé in the cortex were greatly shrunken and contracted, their processes being indefinite and altered in direction.

The patient had been a healthy child till his second or third year, after which the disease had gradually developed. The first symptom was an occasional slight reel or stagger in his gait. About the age of six or seven this reeling became more marked, and almost constant. He was first seen by Dr. Fraser when about twenty years of age. At that time he was employed delivering newspapers. He walked with the gait of a very drunken man, reeling greatly, his body inclined forward, his head thrown back, apparently in constant danger of falling on his face. He was obliged to give up his occupation when about twenty-seven years of age, and was afterwards often seen running forward, then catching hold of a lamp-post, and then again going on. Although he constantly swayed about, there is no mention of giddiness in this case.

In the sister of this patient similar symptoms existed. She was unable to write in consequence of the want of power to make the necessary movements. She could not walk for fear of falling forward. Her disease had slowly advanced as the brother's did. She also was not usually affected with giddiness, indeed it was seldom a prominent symptom, except when she lay down. She then became giddy, or her sight grew indistinct. If she turned her head the feeling became exaggerated, and often she had visceral sensations, such

as a feeling of nausea, but she did not vomit. It is certainly reasonable to conclude that in the female patient the lesion was the same as existed in the brother's case.

Here, then, under certain conditions, severe vertigo occurred, while usually it was not present, and apparently it was not the cause of her reeling.

Ferrier states that division of the cerebellum down the middle line, and lesions which are symmetrical on both sides, do not cause more than slight disturbance of equilibration. Professor Schiff once showed me in Geneva two dogs on which he had performed cerebellar section. In the one, the gait was very staggering; in the other, the staggering was slight; and he told me that in the former he had injured one lateral lobe, in the latter the two lobes equally.

Ferrier has shown that where the anterior part of the middle lobe is injured, the animal tends to fall forward on his face. When the posterior part of the median lobe is injured, the head is drawn backwards, and there is a constant tendency to fall in that direction. Ferrier satisfied himself on these points by experiments on a monkey, and clinical observations have confirmed his experimental results.

### (*b*) *Crura Cerebelli*

In lesions of the crura cerebelli giddiness often occurs, and the patient shows a tendency to fall towards one particular side. In illustration of this, I may cite a case recorded by Friedberg and quoted by Nothnagel in his admirable work on the Localisation of Brain Disease. A patient of six-and-twenty was, on 5th October 1855, struck on the forehead, and fell backwards upon the pavement, but was able to walk home.

Fracture, with depression of the right parietal bone was found. On 15th October symptoms of meningitis set in; the skull was trephined four weeks afterwards; the patient began to go out, and was quite well and able to work until August 1856. Then he began to have occasional headaches, and they gradually became more severe. In October attacks of giddiness, often attended by vomiting, came on. On the morning of the 24th of that month, when he got up and was attempting to walk to the washhand-stand, violent giddiness occurred, and he was turned irresistibly to the left side, so that he could only make his way to the stand moving tumultuously, and in a half circle concave to the right. Next night he was again seized with intolerable giddiness, and suddenly began to turn round in bed on his long axis, from left to right. This was repeated five times. In the course of the same day three similar attacks occurred; first the head was turned to the right, then the thorax, then the under part of the body. When the body had reached the right side, the rotation went steadily on, the extremities sometimes followed voluntarily, sometimes seemed to strive against the movement. The eyes wandered anxiously here and there, seeking help. A complete attack lasted only a couple of minutes, as it then subsided. The patient continued to have frequent attacks of giddiness, and the feeling as if he were about to fall to the right, and as if it were necessary to support himself. On 21st November he took a feverish turn, and his right leg and arm became paralysed. On 30th November he died. The autopsy showed the cerebrum natural, the left half of the tentorium cerebelli prominent. Under the left lobe of the cerebellum there was a rather fresh bloodclot of about fifteen grammes. There was fracture of the right part of the occipital bone. There was exudative

meningitis round the left lobe of the cerebellum, with red softening of its cortical substance; the inflammation of the pia mater extended along the left cerebellar peduncle to the pons, close down to that structure. The tissue of the peduncle was in a state of red softening, with small extravasations to a depth of from one and a half to two lines; the other parts of the cerebellum, pons, and medulla oblongata were natural. In this case it is reasonable to assume that the giddiness resulted partly from the cerebellar, but probably also from the peduncular disease; the peculiar movements were characteristic of the lesion in the peduncles.

I shall cite another case which has been quoted by many writers, and was originally recorded by Belhomme. A lady of sixty had long suffered from giddiness and weakness of the legs, but was seized one day with an attack which lasted half an hour, in the course of which she was constantly constrained to turn round. A fresh attack followed upon great mental excitement. This time the tendency was to turn to the right. The attacks became more frequent, till at last they occurred four or five times in a day. The condition became gradually worse; the attacks assumed a pretty definite character: the patient suddenly lost consciousness, and drew herself together spasmodically; she turned herself with astonishing rapidity, usually to the right, but sometimes to the left. She died of bronchitis nearly ten years after the commencement of the illness. On examination, it was found that the middle cerebellar peduncles both showed a depression, the left more marked than the right, due to growth connected with the bone. On section through the pons, there appeared between the middle and posterior thirds a varicose injection of crescentic shape, the pons itself was one-third smaller than natural; the walls of the fourth ventricle

softened and disorganised. In this case you observe that the symptom of giddiness had existed for more than ten years, and considering the character of the changes met with after death, it is reasonable to suppose that it was due to the pressure upon the cerebellar peduncles, although it may be maintained that they were referable to the cerebellum itself.

The explanation of the mechanism of these phenomena is not yet clear, although it seems pretty certain that lesion or section of the superior cerebellar peduncles cuts lines of afferent and perhaps also of efferent impulse.

Dr. Hughlings Jackson has advanced the theory that the reeling gait of cerebellar disease is due in part at least to weakness of the spinal muscles, whereby the legs, as it were, run after the trunk, striving to balance it as it sways about. In support of this view, observations upon cases of cerebellar disease have recently shown that the giddiness of such patients on walking can often be greatly diminished or even entirely abolished when the weight of the trunk is taken off by supporting the axillæ. One such patient could not walk a step without the most intense giddiness, whereas when supported in this manner the giddiness at once ceased, and she exclaimed that she "felt as if she could walk for miles."

Not only will axillary support relieve these patients, but support of the occiput or even pressure on the occiput may suffice to do so, but in any case the favourable effect must be wrought by diminishing the unsteadiness, and the relief of the sensation of discomfort or the actual giddiness is to be explained on the principle already referred to, that sensory alterations habitually associated with motor changes are sometimes coincidently induced when the motor changes are developed.

With regard to the staggering and giddiness symptoms in cerebellar disease, I may mention certain facts re-

garding cases of which I have, along with Dr. Gibson, published an account in the Edinburgh Hospital Reports for 1897.

The first case was that of a woman from whom Professor Annandale removed a tumour situated in the right lobe of the cerebellum. She complained much of giddiness when she walked or stood upright, or even when she moved in bed, but was quite free from it so long as she lay still. Both the staggering and the giddiness were caused by the operation.

In the second case a man had all the symptoms of cerebellar disease, but declined to submit to the operation which I recommended. He stated that he always felt giddy, but that it became worse when he stood up, and still more if he lent forward. His staggering was distinct when he stood up, or attempted to walk. But although giddy he scarcely staggered at all if he walked slowly with his eyes fixed on the ground.

The third case was one in which operation had been decided on, but unfortunately death occurred suddenly from failure of respiration before the operation had been carried out. She complained greatly of giddiness, and staggered when she attempted to walk, swaying even when she tried to stand.

The fourth case was that of a man who suffered from cerebellar disease along with other intracranial troubles, and who was operated upon but only with temporary success. His giddiness was so severe that he was unable to walk or even to stand without support.

The fifth case was that of a groom aged forty-six. He complained much of giddiness, which was indeed often so severe as to make him fall, but he also at times staggered and fell backwards, not as a result of giddiness but apparently quite independently of any such sensation. In this case Professor Chiene operated successfully,

and both the giddiness and the staggering disappeared.

Such cases as these have satisfied me that disease of the cerebellum may give rise to giddiness, or to staggering, and that either may predominate, and that each may induce or aggravate the other. I do not think that the staggering is always a result of weakness of spinal muscles, for that weakness is not always present, but beyond the general statement that equilibration is a function of the cerebellum, it cannot be said that we have a satisfactory explanation.

It is difficult to understand the relationship between disease of the cerebellum and giddiness; but it seems certain that the greater part of each lateral lobe stands apart from that symptom, and that it occurs only when the middle lobe and the fibres which connect it with the restiform body and the superior cerebellar peduncle are involved. These fibres appear to be mainly devoted to the maintenance of the equilibrium. They are lines along which afferent impressions are conducted of which we are not conscious, but which to the nerve centres, although not the mind, convey important guiding impressions.

## LECTURE III

Gentlemen,—At the end of last lecture I was about to speak of giddiness as a symptom in cases of disease of the pons Varolii. But before doing so I wish to demonstrate a typical case of cerebellar disease, which I am able to show you by the kindness of Professor Fraser.

You observe that, even as he stands, this patient sways about in an irregular manner. Now, when he attempts to walk he staggers like a person in a state of intoxication, and even when I make him sit down, you perceive that he seems inclined to fall in one direction or another, and then to catch himself by a sudden effort. He tells me that he constantly feels giddy, but I find that his giddiness is increased by his movements. If the back of his head is pressed against the wall, or if it is held firmly, his giddiness almost, but not altogether, disappears. It is thus, then, in some degree, a consequence of the movements, but it is not entirely dependent upon them. It is due in part to some sensory change.

### *From Disease of Pons*

And now, to return to the pons Varolii,—I shall cite in illustration one or two cases. Gnéniot describes the case of a woman forty-seven years of age, who was suddenly seized with giddiness, with loss of power of speech, and paralysis of the left arm and leg. These

conditions persisted. The tongue was paralysed, but there was no difficulty in swallowing; intelligence was good, and there was no affection of the facial muscles or those of the eye. Sensibility was retained in the paralysed parts—apparently rather increased; death followed in four days. On examination, it was found that the brain was natural, but the pons broken up by clot. On closer examination of the specimen it was found that while the right pyramidal bundle was completely destroyed, the left was scarcely affected. The point of interest for us in this case is the giddiness. It was manifestly a prominent feature, and it was not the result of paralysis of any of the muscles of the eye. No cause was found excepting the lesion of the pons, and to it the well-marked vertigo must manifestly be ascribed.

Another case may be cited, which was observed and recorded by Professor Nothnagel. A syphilitic man of forty-six was seized with headache and giddiness, with tendency to fall to the left side, and loss of consciousness. After two or three attacks of that kind, he was admitted to hospital, a permanent condition having become established; there was no paralysis of face, eye, or tongue, and speech was normal; but there was complete motor paralysis of left arm, and paralysis of left leg. Sensibility was unaffected. The reflexes could not be produced in the left arm. For two months there was no change, and in particular no tendency to muscular contracture. Death resulted from pneumonia; and on examination, it was found that a mass of softening, of the size of a hazel nut, was situated on the upper half of the right side of the pons, near the cerebral peduncle. In this case, also, you observe that giddiness was a prominent symptom, and that softening was the only change found on examination.

It being clear, then, that lesions of the pons itself

are capable of producing giddiness, even when double vision is absent, it is scarcely necessary to adduce proof of it resulting in cases where squinting occurs. But such cases have also been observed.

When we consider the importance of the superior olivary body as a centre dealing with sound impressions and with the regulation of equilibration and its manifold connections with other nuclei, we may well understand how, even apart from squinting, giddiness may arise when it is injured.

### *Cerebral Peduncles*

In lesions of the cerebral peduncles giddiness is sometimes seen, but usually it results from squinting due to paralysis of some of the muscles of the eye. Such a case as that which I am about to state, suggests that other influences may operate here. It is recorded by Rosenthal and quoted by Nothnagel. A woman of thirty-nine had suffered for two years from headache, giddiness, and dimness of sight, with paralysis of the right side. The giddiness was so great that the patient preferred the recumbent position. Severe giddiness set in if, even when recumbent, she turned on her left side. She had paralysis of the third left cranial nerve with paralysis of the lower branches of the facial and of the limbs; on the right, distinct anæsthesia. After death, it was found that a tumour of the size of a pea lay between the crura cerebri. The innermost part of the right cerebral peduncle was softened. In the interior of the left crus cerebri there was a cyst of the size of a pea, which destroyed the left third nerve, and another tumour of the size of a bean involved the right oculomotor.

I think you will agree with me in believing that, although the squinting may have been a factor in the

production of giddiness, there certainly was a further influence in operation, seeing that the symptom was so severe.

The paralysis of the third left cranial nerve could not explain such a giddiness as this, and considering that the red nucleus which is the upper termination of the superior cerebellar peduncle lies in intimate relation to the third nerve, it seems certain that that structure must have been involved by the small cyst. The mechanism underlying the giddiness is thus rendered clear.

### *Corpora Quadrigemina*

In lesions of the corpora quadrigemina giddiness sometimes occurs, at all events staggering gait has been observed in some cases. In illustration of giddiness from this lesion, I select a case recorded by my friend Dr. Duffin, of King's College, London. The patient was a steady and powerful man, and the earliest symptom—a sense of dragging at the back of the neck—occurred about four months before his death. The walk early became unsteady, and there were repeated attacks of vertigo, which diminished when the eyes were closed. Headache and contraction of the muscles of the neck and spine were prominent symptoms. Sight soon became impaired, and blindness speedily followed with well-marked double optic neuritis. The intelligence became obscured only about a fortnight before death. He died comatose. The autopsy showed the corpora quadrigemina and the pineal body replaced by a glioma. The right optic thalamus had atrophied from pressure, and the right superior peduncle of the cerebellum was also involved. It is possible that the giddiness was connected with change in the peduncle, but considering that the tumour involved primarily the corpora quadrigemina, and

the pineal gland, and that the giddiness was an early symptom, it is more reasonable to refer the giddiness to the altered condition of the corpora quadrigemina themselves.

But in any case the fibres of the lateral fillet which pass into the gray matter of both corpora quadrigemina must have been involved. And one of their functions is believed to be connected with equilibration.

### *Undefined Intracranial Causes*

Besides these distinctly localised intracranial causes of giddiness, there are others which seem to act upon the cranial contents generally,—for example, cerebral tumour in any part of the brain may be found to produce occasional fits of giddiness, and alterations of the cerebral circulation induce a like result.

### *Cerebral Congestion*

Congestion, whether of active or passive origin, induces giddiness, sometimes to such an extent as to prevent the patient from walking, and often sufficing to render quick turning round a matter of difficulty. In treating such a condition, one must first advert to the cause. If it be passive congestion, seek to remove the obstruction; if it be the result of plethora, diminish the volume of the blood; if it be from overwork, or other undue excitement of the brain, or from a tendency to inflammation, use such remedies as cold to the head, ergotin, the bromides, and perhaps derivatives.

### *Cerebral Anæmia*

Vertigo is a common symptom in Cerebral Anæmia. It is apt to be induced when the patient suddenly changes

his attitude, either by lying down or getting up. Sometimes it is merely what the patients describe as swimming in the head, at others it is well-defined vertigo. It is often associated with neuralgic and other forms of headache. You will meet with it very frequently in cases of chlorosis. Indeed it is sometimes one of the most troublesome symptoms of that disease. It is also occasionally met with in cases of anæmia due to loss of blood, slight and long continued, as in bleeding hæmorrhoids, or more formidable as in menorrhagia or other uterine complaints. Sometimes, also, it is seen in the later stages of aortic valvular disease, when the cerebral anæmia becomes pronounced; and sometimes in cases of unexplained anæmia, whether referable to the pernicious variety or not, it also occurs. In these cases you will find that the patient suffers less (except when special peculiarities come into play) after food has been taken, or after a little alcohol, and that he is sometimes markedly relieved, for a time at least, by the use of nitrite of amyl. The treatment of the giddiness in such cases is, of course, the treatment proper to the variety of anæmia which may be causing it, and usually consists in checking any drain of blood which may exist, and improving the condition of the blood itself. Let me remark in regard to one of these causes—viz., the bleeding from hæmorrhoids,—that you will often have to deal as physicians with this complaint, and that I know of no plan of treatment more successful than that which I learned from Professor Oppolzer of Vienna—viz., the regular use of aloes and sulphate of iron. If you will direct your patient to take morning and evening, or three times a-day if it be necessary, a pill containing one grain of aqueous extract of aloes, one-third of a grain of sulphate of iron, and two grains of extract of taraxacum, you will find that free soft motions are produced, while the bleeding as a

rule rapidly disappears. In this way your patients may often be relieved without having recourse to surgical treatment. I say nothing of the treatment of the uterine or other hæmorrhages; but should like to accentuate the fact that in the effort to improve the condition of the blood, when our chief remedy, iron, fails, arsenic often stands us in good stead.

Vascular changes of the nature of arterial sclerosis often induce giddiness under various circumstances, but especially in sudden change of position, owing to the lesion of the vessel walls rendering them incapable of accommodating themselves to varying conditions of blood pressure.

### *Nervous Exhaustion*

Vertigo is sometimes due to mere exhaustion of the nervous system, as from overwork, sexual excesses, and such like; this occurs quite apart from anæmia, is manifestly a result of changes in the nerve centres themselves, and is to be treated by means of avoidance of the cause, rest, good diet, and nervine tonics—chalybeates often proving unsuitable.

### *Giddiness from Injuries to Head*

Giddiness sometimes results from and persists for long after injuries to the head. The patient is thrown from horseback or from a bicycle, and, falling on the head, is for a time stunned; on recovering, he is at first much troubled with giddiness or with headache, and he finds that for months, or for life, he is much more easily made giddy, becoming so under conditions in which previously no giddiness would have occurred. The nervous system has acquired a new mobility of this and of other kinds. You will find that patients who have

suffered from sunstroke, even to a slight extent, often exhibit the same peculiarity.

### *Associated with Epilepsy*

A very distressing form of vertigo is that which is associated with epilepsy. One often meets with such cases in practice. Take as illustration a case regarding which I was recently consulted. A lady, aged forty, sustained an injury to the head about seven years ago. From that time she became subject to attacks, usually merely of giddiness, but occasionally of complete unconsciousness. On examination, nothing was found in the state of the ear or of the alimentary tract to account for the condition, and the only satisfactory explanation was that which referred the process to the category of epilepsy. Such a diagnosis as this can scarcely, of course, be established in the absence of more typical epileptic attacks. The treatment in such a case must consist of careful regulation of diet, the administration of bromides and belladonna, and, in rare cases, operative interference at the seat of cranial injury. You must also note that the epileptic may have giddiness as a warning at the onset of his attack, a variety of aura epileptica.

### *Associated with Migraine*

In other nervous affections, such as migraine, giddiness occurs. It is not in my experience a frequent symptom, but is met with occasionally towards the end of the first stage, when the ocular or other sensory troubles are nearly passed, and the headache is about to commence. Sometimes it occurs later, when the headache is fairly developed, and the nausea or vomiting has come on. Sir William Gowers has pointed out that it sometimes occurs

as the only manifestation of a paroxysm of migraine. The diagnosis of the cause of the form of vertigo is easy to any one who is acquainted with the features of migraine. The treatment in such a case is of course that applicable to migraine generally; and that again is guided by a study of the cause, whether it be connected with gastric and hepatic derangements, with constipation, or with nervous exhaustion. The mention of each of these causes will suggest to our minds the appropriate remedies; but, in addition, you will find that, in many cases, the attacks may be warded off by the regular use of the bromides, or of guarana, or chloride of ammonium, or of an alcoholic stimulant, or antipyrin, antifebrin, salophen or caffein, or even a cup of tea or coffee.

### *Periodic Giddiness*

As an illustration of a rare but very interesting form of vertigo which we may call Periodic Giddiness, I select a case which was under the observation of my late assistant, Dr. Purves Stewart, when he was resident in the National Hospital for the paralysed and epileptic. The patient, a boy of fourteen, had suffered from the age of seven from periodic attacks of giddiness and headache, recurring with great regularity every eighty or eighty-one days (eleven and a half weeks). His parents were unable to suggest any cause for the attacks. At the beginning of his illness the attacks used to last five days. When he had reached the age of nine each lasted seven days, and at a later date they had extended to eleven days as a rule. But he always had eleven weeks of absolute freedom from his illness, and during these intervals was in perfect health. As to the attacks themselves the great features were giddiness, frontal headache, and latterly diplopia.

The giddiness came first, the headache followed in about a couple of days. The two then went on increasing till about the fifth day, then gradually subsided. The patient was carefully observed during two of his attacks, one commencing 10th December 1896, and the other 3rd March 1897. He lay in bed on his right side with the thighs and the knees flexed, and complained of giddiness and frontal headache. When he was asked to stand he was very unsteady, and staggered in attempting to walk. He spasmodically twisted and rotated his head, holding his head between his hands with the view, as he explained, of relieving the giddiness. In regard to his eye symptoms, the only peculiarities were divergence with rotation upwards and crossed diplopia. But there was no actual ocular palsy, the eyes could be moved voluntarily in any direction. When he was made to look down the giddiness was much increased. Most interesting of all was the fact that when standing his giddiness was entirely removed if he closed his eyes. From these circumstances I conclude that the vertigo in this case depended upon disturbance of the ocular muscles, but I can offer no explanation of the periodicity of the attacks. Otherwise the condition links itself on to what Sir William Gowers describes as epileptoid vertigo, at least it presents several features in common with what he has described under that name.

### *Giddiness from Degenerative Arteries*

Giddiness is often complained of by old people, particularly those affected with degenerative changes in the arteries, and in such cases is a cause of much alarm, being popularly and justly associated with the idea of a tendency to apoplexy. Whatever may be the precise pathology of this variety, I would have you bear in mind

that one very able physician, Dr. Handfield Jones, has found the bichloride of mercury, in small doses, of very great service in such cases.

### *Giddiness from Electric Current*

There is a form of intracranial giddiness which you can easily experience experimentally for yourselves,—viz. that described by the late Professor Purkinjé of Prague, as being produced when a constant current of sufficient strength is transmitted through the head from ear to ear. If you place the electrodes in the mastoid fossæ, and allow a current of medium strength, say ten or twelve cells, to pass, you will find immediately that giddiness results, external objects appearing to rotate in the direction of the current, while you incline to turn in the opposite direction. It requires a much stronger current to bring out movements of the eyeballs, such as Hitzig has described. It may turn out that these changes are due to an influence exerted on the semi-circular canals; but this seems improbable, both from the locality in which the electrodes have to be placed, and from the occurrence of the nystagmus when stronger currents are employed.

### *Diagnosis and Treatment of Giddiness of Intracranial Origin*

With regard to the diagnosis in cases of giddiness from intracranial disease, I should advise you to satisfy yourselves first as to whether there is paralysis of any of the eye muscles or not, and not to conclude that giddiness is due to brain lesion unless other and more characteristic symptoms of intracranial disease be present.

The treatment of giddiness from intracranial causes, other than those already referred to, is not very promising at present. There are some cases in which the lesion being syphilitic, iodide of potassium, or mercury, or a combination of the two, are of great service; there are probably some in which the bichloride of mercury is useful, even when the disease is not due to syphilis; but on the whole, medicine is of little service. Now and then a surgical operation does good by removing some tumour or irritating fragment of bone, or by diminishing intracranial tension.

### *Giddiness from Gastric Disturbance*

Having thus traced the various nervous causes of vertigo with which we were acquainted, I now proceed to describe a number of others which you will find come frequently into operation. Among those we shall take first dyspeptic giddiness. It is matter of common observation that, along with gastric and hepatic disturbance, headache and giddiness frequently occur. But there is in addition a formidable and alarming kind of giddiness, which has been well described by Trousseau in his clinical lectures, and by Dr. Ramskill in "Reynolds' System of Medicine." It is known by the name of stomachal vertigo, or *vertigo a stomacho læso.* The case generally presents such features as the following: The patient, an hour or two after taking food, is subjected to some fatigue, excitement, or emotion, and suddenly becomes very giddy. He reels, perhaps falls to the ground, becomes sick, may lose consciousness, or may simply suffer from a peculiar headache. He seems to himself to be turning over in all directions, or to have currents within his head, which are flowing, in a curved direction, from before backwards, or from behind forwards,

or in an irregular manner. Surrounding objects seem also changed. The ground feels to his feet like the deck of a ship on a stormy sea, while other things seem to be constantly changing their position, swinging with jerks upwards or downwards, backwards or forwards; and while he knows that these are illusions, he yet cannot steady himself. Perhaps vomiting takes place; perhaps there is a copious discharge of flatus from the stomach or the bowels; perhaps sudden diarrhœa. Then he becomes almost well, the giddiness passing off, and he is free from the illness for the time. Now, it may be that you find—as Dr. Ramskill found in one of his cases—that the patient has had a hearty breakfast, including sausages and Devonshire cream, or some other things equally difficult of digestion, or the diet may have been perfectly simple. One attack seems to render the patient more liable to the recurrence, and your patient may give you the history of many seizures of varying severity.

This form of vertigo may be transient, or may be persistent for a time. Some years ago I was consulted by a lady whose home was in a distant part of Scotland. She was little and stout, and came into the room with a curious dazed expression, putting her hand on a chair or a table at every opportunity for support, and still occasionally reeling a little like a person on board ship who has not acquired his sea-legs. She stated that life was made intolerable to her by various uneasy feelings, the billowy rolling of the floor being the most distressing; but that occasionally she had fits of unconsciousness, and sometimes convulsions. Her tongue was furred; her appetite was impaired; and she suffered from constipation. There was no deafness nor noises in the ears, nor any other condition characteristic of Menière's disease. Neither was there any fixed pain in the head,

nor eye changes, paralysis, or convulsions, such as might have been indicated by cerebral tumour, and her illness had already lasted for years. I was satisfied, from the result of treatment, as well as afterwards from the absence of important symptoms, that this case was to a large extent dyspeptic in its origin, the dyspepsia being implanted upon a very sensitive nervous system. The fits of unconsciousness I regarded as syncopal, the convulsions as probably hysterical. When she was treated by means of stomachics and aperients, with regulation of the diet, she became greatly better, and now enjoys fairly good health.

A patient lately presented himself to me much alarmed about attacks of vertigo. He was somewhat advanced in years, of large frame, and had retired from professional work, which had, he told me, been laborious. As he suffered much from feelings of debility, he took food at short intervals and in considerable quantity; being unfit for active bodily exertion, he tended to grow stout. Gradually he became subject to attacks of giddiness, associated with extreme anxiety, dimness of vision, and nausea, and even vomiting. The vomiting was attended by partial relief, and was followed after an hour or two by complete recovery. In this case also there was nothing characteristic of labyrinthine or cerebral disease, and I confidently anticipate, that with the establishment of better habits, as to diet, and regimen, and with the regular use of stomachic and aperient remedies, he will quite recover.

In the treatment of dyspeptic vertigo, Trousseau had the utmost confidence in the use of alkalies and bitters; he used to give a dose of quassia every morning and at bedtime, and after meals a powder containing 5 grains of bicarbonate of soda and 5 grains of magnesia with 10 grains of prepared chalk, the powders were occasionally

alternated with alkaline mineral waters, but a not unimportant part of the treatment was the insistence on careful dieting and regular bodily exercise.

Remember, then, that as to diagnosis, the hints that I have given you will guide you, and that in the treatment of this form of giddiness you will often effect a cure, and constantly obtain great relief for your patient by very simple means. Regulate the diet as to quality, quantity, and times of eating. Give aperients if they are required, alkalies, and bitters. Give stomachic remedies, such as rhubarb, bismuth, soda, and aromatic powder, followed by nux vomica, quinine, or calumba or chiretta, and the good effects will speedily manifest themselves. When there is much flatus in the stomach, carminatives often do good, and if the ordinary ones fail you will find that from ten to fifteen drops of turpentine may give great relief.

Now, what is the nature of this form of vertigo? Is it reflex? Is it a result of alterations of circulation within the brain? Is it a result of poisoning? It is impossible, in the present state of our knowledge, to answer these questions definitely, but I lean to the opinion that it is of the nature of poisoning, that in connection with the chemical changes of the digestive process under certain conditions, some substance is developed which, like alcohol or tobacco, acts upon the nerve centres so as to induce the peculiar uneasiness.

### *Toxic Vertigo*

Among the commonest forms of vertigo must be reckoned the toxic, and among them those due to alcohol and tobacco. When the action of alcohol has reached a certain point giddiness is experienced, and associated with this is the reeling gait. Objects may

appear to whirl round the patient, or he may appear to be rotating—the movement may seem to be irregular, or in certain definite directions, as from right to left, or from left to right. You know the case of a well-known legal luminary in bygone days, who was found by a friend standing rather unsteadily in the square in which he resided, making no progress towards his door. When interrogated as to his object, "Don't you see," said he, "that the square is going round and round, and whenever my door passes I'm going to jump in." Or the apparent movement may be a rotation on a horizontal axis, as in the case of the man who was found lying on the ground, and who explained that the earth was turning rapidly round, and he was holding on by the grass to prevent it. We may not be in a position at present to state positively how this giddiness is brought about, whether through alterations of the vascular system, or by direct influence upon nerve centres, but the latter seems to be the more probable explanation. The effect of tobacco on those unaccustomed to its use is similar to that of alcohol in the production of giddiness; and other toxic agents might be mentioned which induce a like result—such as stramonium, belladonna, hydrocyanic acid, lobelia, hyoscyamus, and veratrum viride.

Occupying an intermediate position between the toxic and the dyspeptic, and perhaps also related in some measure to the labyrinthine vertigo, is the variety occasionally met with in gouty patients, sometimes slight, sometimes really severe, and which is apt to occur in the intervals between regular attacks of gout, especially as the attacks draw near, and which is often absent for a considerable time after the typical seizure. This form is, in my opinion, often referable to the stomach, but it may also, perhaps, be induced by toxic influence upon the nerve centres.

Besides these obviously toxic forms of vertigo, there are others which are reasonably to be referred to the same category—I mean the giddiness occurring in acute specific diseases. In the commencement of fevers, the symptom is not uncommon—just as we have commonly rigors and headache, and occasionally convulsions, as initial phenomena. It has been remarked that in influenza it is frequently very troublesome;—sometimes as one of the earliest indications of morbid action; sometimes during the height of the disease; and that occasionally it continues for long or even originates after the acute stage of the malady has passed. It bears the same relationship to many acute diseases. In some of these cases, it is probable that at first a direct toxic influence is exerted, while at a later stage exhaustion or actual lesion of the nerve centres or nerves may be regarded as the most likely cause. In the case of influenza, the same set of causes may be supposed to come into play, although it may be reasonably assumed that catarrh of the Eustachian tubes accounts for it, in some instances. In such cases it would be attended by more or less impairment of hearing. In respect of treatment you will do well to keep the patient at rest and in the recumbent position, as free as may be from the exciting causes of giddiness; for you will find that sitting up or attempting to read will often suffice to induce an attack. During the later stages, tonics and nutrients, with moderate doses of alcohol, prove most serviceable.

### *Vertigo from Coughing, Sneezing, and Laryngeal Neurosis*

Vertigo is sometimes produced by violent fits of coughing; sometimes by violent fits of sneezing, but by

far the most interesting cause of giddiness due to changes in the respiratory functions is that described by Charcot as Laryngeal Vertigo. Dr. M'Bride has written able papers on the subject, and done much to clear up both its clinical history and its physiology. He points out that "in every case the fit was preceded by a short cough or in other words by a series of spasmodic inspirations, followed by spasmodic expirations with partially closed glottis." If complete spasm of the glottis occurs after a full inspiration the condition within the chest must correspond to that during the act of coughing. He believes that the attacks are due to this, and are brought about by a more or less complete attack of syncope resulting from the pressure of the air which fills the chest and cannot find exit in consequence of the spasm of the glottis. This seems to me a better explanation than that which referred it to possible alterations in the condition of the middle ear.

### *Vertigo from Peripheral Irritation*

In some cases giddiness is due to peripheral irritation, and that irritation may be situated in various parts of the body. As a typical example, I select the giddiness which sometimes results from the presence of worms within the alimentary system. You are aware that other nervous symptoms, such as convulsion and cough, arise from this cause, and we may assume that the vertigo is brought about in the same way as these other symptoms. It is most reasonable to refer them, in the present state of our knowledge, to reflex action, and in the case of giddiness, to an influence upon the cerebral circulation. The treatment in such a case is, of course, obvious. Let the irritating cause be removed from the intestine, and the giddiness will disappear.

### *Vertigo from Mental Causes*

Vertigo arises in some individuals from purely mental causes. A patient goes on board a steamer, and although there is no tossing, and no perceptible vibration, the recollection of what he has suffered before, or the sight of the oscillation of the waves, makes his head swim or renders him actually giddy. In some, giddiness supervenes if a child is seen whirling rapidly round. I have known it even produced by another person walking in a small circle. The very thought of giddiness suffices in some to induce the sensation, and almost every one of the varieties I have described is liable to be aggravated under mental influences.

### Conclusion

We have thus passed in review the chief causes of this sensation. We have considered giddiness as arising—

(*a*) From external conditions.
(*b*) From changes in the sensory structures.
(*c*) From changes in the conducting fibres.
(*d*) From changes in the central nervous system.
(*e*) From dyspepsia.
(*f*) From toxic causes.
(*g*) From respiratory disturbance.
(*h*) From peripheral irritation.
(*i*) From mental causes.

By following out the indications which I have given, you will generally be able to determine with certainty the particular cause in any individual case, and thereby be able to devise a rational treatment.

# STUDY OF A CASE OF HYSTERIA IN THE MALE, EXHIBITING SOME FEATURES NOT HITHERTO DESCRIBED

## History and Description of Case

W. F. is a tall, spare man, æt. 49, who says that he was formerly a belt cutter, and afterwards a commercial traveller; that he has often been ill, having had Bright's disease, prolapse of the rectum, headache, palpitation, breathlessness, and a great variety of other maladies. He was sent to the Royal Infirmary, 20th August 1893, as a case of Bright's disease, but the albuminuria soon disappeared, and the œdema, which was distinct on admission, passed rapidly away. But as they passed off various nervous symptoms manifested themselves, and these have continued with little alteration during the five months that we have had him under observation.

His family history is unimportant, except that his father died of brain disease, apparently apoplexy, at the age of 58. His social surroundings were said to be favourable, but exposed him a good deal to changes of weather, and he seems to have indulged in alcohol to a considerable extent. He says that he has suffered much from prolapsus ani, and has undergone an operation for its relief. He also describes an attack of convulsions which occurred shortly before his admission to the wards. He asserts that the doctor who then attended him regarded them as uræmic. The amount of Bright's disease

that we witnessed was certainly not such as should have led to anything of the kind, and I incline to think that the seizures must have been of a purely functional nature.

He is a tall, fair man, of neuro-sanguine constitution, has an alert manner, and had, especially in the early months of his stay in hospital, an expression of countenance fitted to suggest the propriety of close inquiry into the accuracy of his statements. Latterly his expression has become more one of anxiety and debility. His right foot and leg are constantly in rhythmical motion, except when he is asleep; his left foot and left hand also jerk, but less rhythmically and severely. His eyes show a distinct degree of staring, and the size of his thyroid gland varies remarkably. His temperature is almost always normal.

The *alimentary system* shows nothing of importance except some dyspepsia and considerable diarrhœa associated with the prolapse already mentioned.

The *hæmopoietic system* shows normal blood in respect to the corpuscles and hæmoglobin, normal lymphatic glands and spleen, but the thyroid gland is sometimes of natural size, sometimes considerably enlarged.

With regard to the *circulatory system*, he complains of palpitation, a feeling of faintness, with irregularity of heart's action, but unattended by murmurs. His pulse is sharp, 104 per minute, with very low tension between the beats. There is no thickening of the vessel walls. Pulsation is very markedly felt throughout the whole arterial system, but especially in the abdominal aorta.

The *respiratory system* presents no abnormality, except that the voice is somewhat hoarse, and the expiration is at times much prolonged, apparently from hindrance to expiration.

The *integumentary system* is natural, but for profuse

perspiration, a slight tendency to acne, and an undue irritability of the vessels, which readily produces the phenomenon of factitious urticaria.

The *urinary system* is now normal.

The functions of the *reproductive system* are stated to have been in abeyance for two years and a half.

The *nervous system* shows many abnormalities in respect of its *sensory functions*. There are no important subjective phenomena, nor are there any objective abnormalities on the right half of the body. On the left half all kinds of skin impressions are perceived readily, and with normal acuteness, but they are incorrectly localised. This faulty localisation is constant, and does not vary. For example, an impression of touch, of pain, of temperature, applied, say, over the middle third of the radius, is perceived as if over the middle third of the ulna. If on the thumb, it is felt as if on the little finger, and, similarly, displacement occurs, but always to the same spot over the greater part of the arm and the leg. The left half of the trunk shows a corresponding peculiarity, but above the level of the clavicle there is nothing wrong. In the arm and leg, however, there are areas in which correct localisation exists. Fig. 1 represents the back, Fig. 2 the front of the hand and arm, the dark shaded parts showing the areas of correct localisation, the rest of the limb being abnormal, so that a touch at *A* is felt as if it were at *a*, a touch at *a* as if it were at *A*. So with *B*, *C*, *D*, *E*, with their respective Greek letters, while at *F*, *G*, and *H* there is no displacement. It will be observed in the drawing that in the middle of the front of the forearm there is a small portion of skin stretching across the normal area, in which faulty localisation occurs. Fig. 3 represents the front of the leg, Fig. 4 the back. An impression at *A* is felt at *α*, and *vice versâ*, one at *B* is felt at *β*, and so on. On the front and on the back of

the leg a narrow area is marked by shading, and over this field localisation is correct. Fig. 5 represents the front of the trunk. Above the clavicle the shading marks an area of normal localisation; all the rest of this

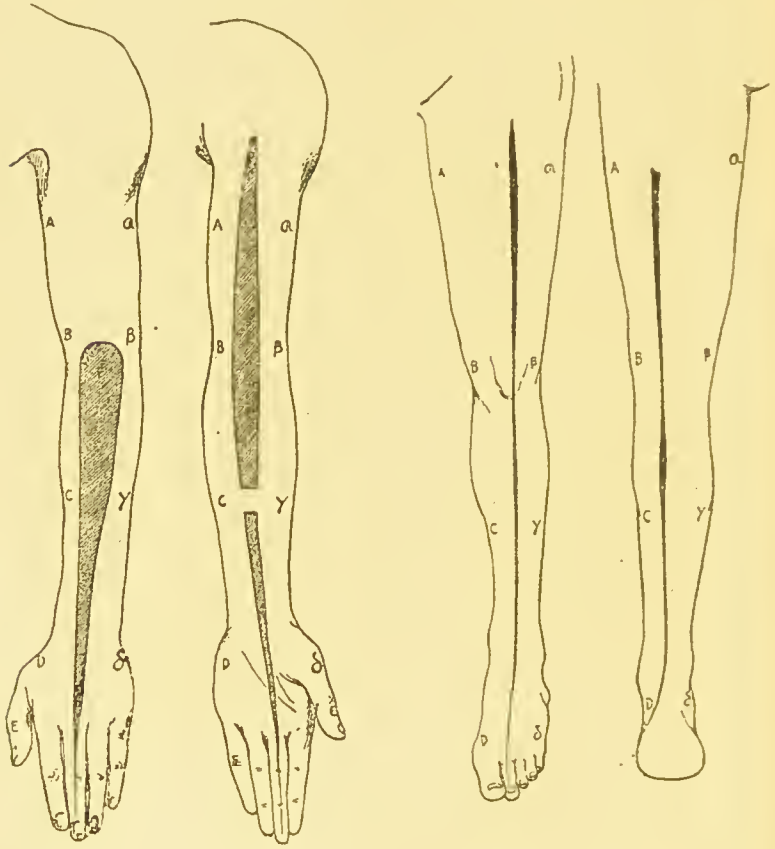


Fig. 1. Fig. 2. Fig. 3. Fig. 4.

part of the trunk shows displacement of impressions except the narrow shaded line, which is normal. A touch at *A* is invariably perceived at *a*, a touch at *B* as if it were at *β*, a touch at *C* as if it were at *γ*, a touch at *D* as if it were at *δ*. Impressions at *E* are referred to *F*, at *F* are referred to *B*, at *G* are referred to *C*, at *H*

are referred to *D*, while at *I*, *J*, and *K* they are correctly interpreted. Fig. 6 represents the back of the trunk. Corresponding to the iliac crest, the shaded area marks normal condition. In all the rest localisation is altered. Thus at *A* a touch is felt as if at $\alpha$; at *B*, as if at $\beta$; at *C*, as if at $\gamma$, and so on. Many separate observations have been made, and the transference has always been in


Fig. 5.

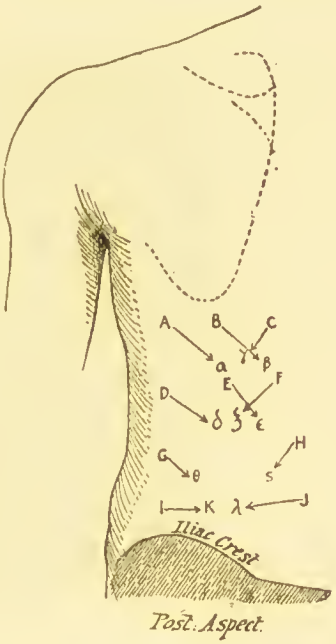


Fig. 6.

the same direction, and to the same points. The whole face and neck, tongue, and lips are normal.

The eyes showed from the first a certain staringness, but this tendency is growing more distinct. Von Graefe's symptom of Graves' disease, the delayed descent of the upper eyelid, was at first present occasionally, but has now become constant. The sight is acute, both as to form and colour; the pupils are equal, and react to light. The patient complains of occasional diplopia, and

in both eyes there is marked contraction of the field of vision. Dr. F. W. Mackay has kindly taken perimetric tracings for us. Fig. 7 shows the field of vision in the right eye as determined on 6th October 1893. It shows in the vertical axis 9° instead of 50° above, and 19° instead of 65° below; in the horizontal axis 10° instead of 60° on the nasal, and 20° instead of 90° on the temporal. Fig. 8 shows that in the left an

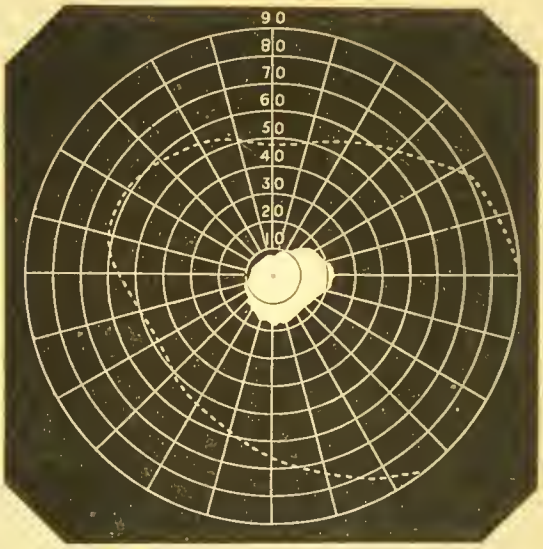


Fig. 7.—Perimetric chart of right eye.

almost corresponding condition exists. A recent examination has shown that this condition remains unchanged.

The field of vision for different colours is altered in an interesting and suggestive way, for the perception of blue is relatively much more diminished than that of red. Fig. 9 shows the result of the observations as to the colour perception. While all are narrowed to some extent, the blue is distinctly less than the red.

The ophthalmoscope reveals no structural change in

the nerve or retina, beyond a degree of pallor of the disc with narrowing of the arteries.

The hearing is deficient in the left ear, but merely from excess of wax. Taste, smell, and muscular sense are normal.

The *motor functions* are disturbed in respect that there is constant movement in the right foot and leg, and this movement is of a rhythmical kind, exactly

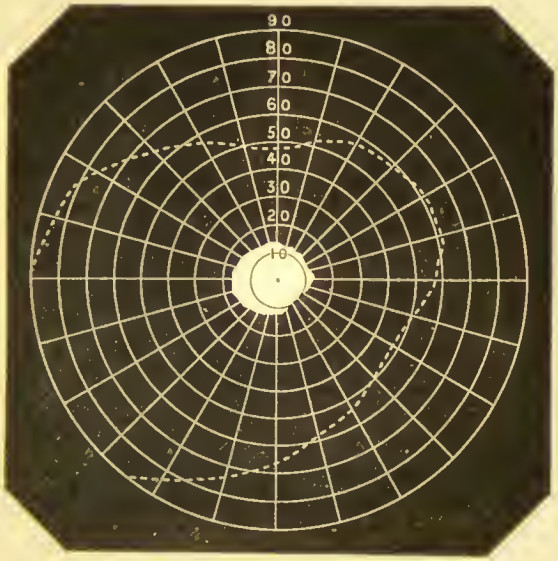


FIG. 8.—Perimetric chart of left eye.

corresponding to ankle clonus. It may be named causeless or spontaneous ankle clonus. Fig. 10 shows a tracing made, with the kind assistance of Dr. James, on 4th November 1893. It shows the clonus of the right foot at the rate of 7 to 8 per second. More recent tracings essentially correspond, excepting in respect of being more rhythmical and regular. In the left foot and in the right hand less rhythmical jerking movements are to be seen; they resemble the movements of chorea rather than the regular oscillation that marks the clonus.

Fig. 11 shows the record of the movements of the left foot at the rate of 5 or 6 per second. As the weeks have gone on, these movements have tended to become more rhythmical. It is to be observed that the motor changes are most marked in the right leg, while the sensory skin changes are on the left side. The organic reflexes are normal, the skin reflexes are well marked,

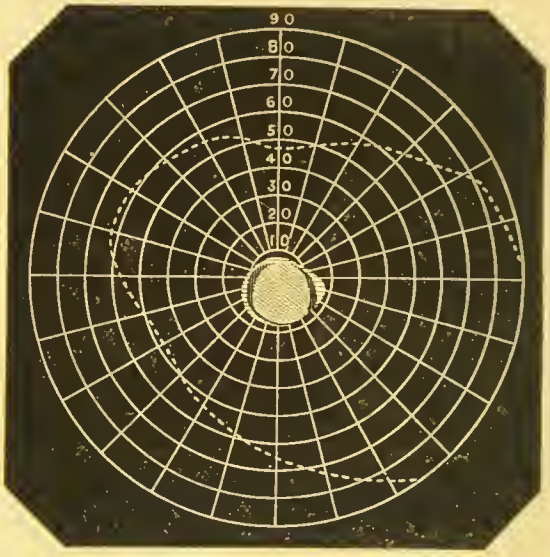


Fig. 9.—Perimetric chart of field of vision for different colours.

indicates green. indicates red. indicates blue.

although their demonstration is interfered with by the clonic spasms. The knee jerk is exaggerated on both sides, and ankle clonus is easily demonstrated in the left foot, less easily in the right, the difficulty being due to the constant jerking. Voluntary movements are well performed, so far as the face, the hands, and arms are concerned. The patient stands as steadily as the clonus permits, and is not made materially worse when he closes his eyes. Fig. 12 shows a taxographic record by Dr.

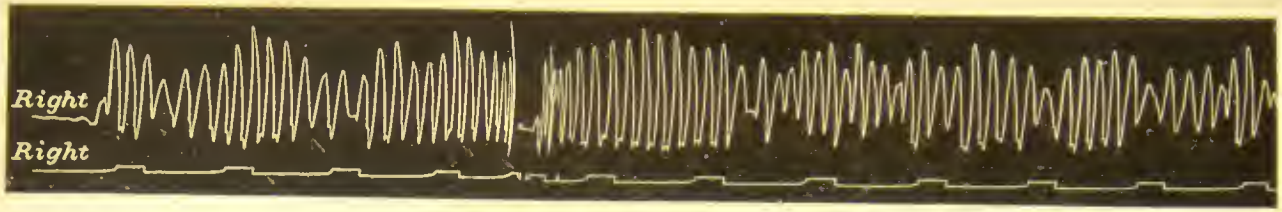


FIG. 10.


FIG. 11.

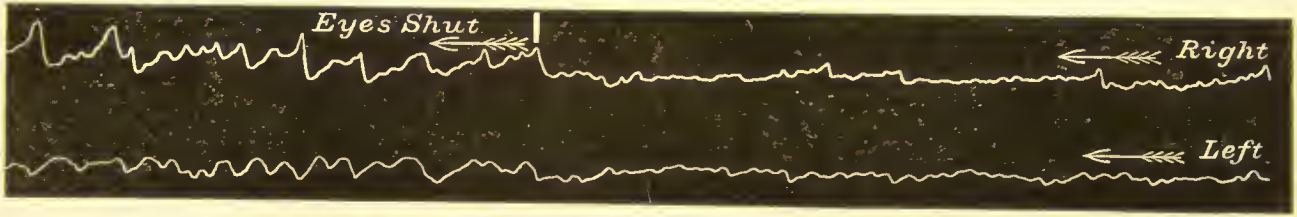


FIG. 12.

James' instrument, which exhibits the process as seen in each foot with eyes open and closed.

His gait is most peculiar, for he advances with a kind of cork-screw movement, taking three steps to the right, then three to the left, three to the right and three to the left with perfect regularity. He turns well enough, and he can walk backwards, but with the same three-step movement. The gait gets worse if he is supported by people on each side of him, and it is brought about by each third step having an exaggerated adduction, so that the foot which is raised for the advance is passed over and in front of the other foot. This disturbs the equilibrium, and the next step is modified so as to correct it. Thus if the left foot has been passed over and in front of the right, he rapidly throws the right one forwards and outwards so as to avoid falling, from loss of equilibrium. As each third step shows the abnormal adduction, the legs suffer alternately, and so the rhythmical cork-screw movement is kept up. He says that it is not the result of any feeling of giddiness, but simply because he cannot help making these movements. Fig. 13 shows a photograph of the footprints taken in Ward 22 by Mr. Godfrey Hudson, clinical clerk, and exhibits more clearly than any description can the peculiarities of the gait. No abnormality has been discovered in the electric conditions of nerves or muscles.

The *vasomotor functions* show, in addition to the undue irritability of vessels, which marks itself by the factitious urticaria, generally distributed throughout the body, an abnormality of the thyroid gland which I have not hitherto seen. On some occasions it is so small that it can scarcely be made out, on others so large as to constitute a distinct tumour. Emotional excitement and physical exercise sometimes cause the enlargement, but

not invariably. The transitions are frequently abrupt, the rise and fall occurring within a few hours; there are no trophic changes. The vascular features have become less marked during recent weeks.

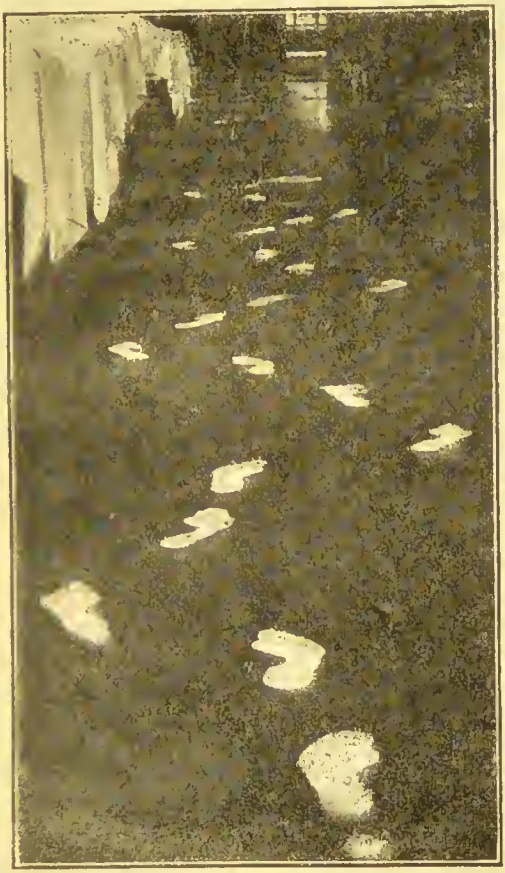

Fig. 13.

The *cerebral and mental functions* were normal at the time of his admission, but have become somewhat modified in the direction of exaltation, such as one sometimes sees in the very early stages of certain cases of general paralysis. The patient sleeps well, and during sleep is

free from his jerking movements, but they recur almost immediately on his awakening.

In regard to treatment I have tried mainly two lines, one by the use of a seton in the back of the neck, with strong assurances that it was likely to do good; the other hypnotism. The patient has never come fairly into the hypnotic state, but seems likely to yield to it if we persevere. The results obtained by treatment have not as yet been striking.

The records of his case have been kept with the most admirable care by the resident physicians and clinical clerks in charge, Drs. Murray Leslie and Cattanach, and Messrs. Laing and Murray Stewart; but I feel especially grateful to Mr. Laing for his unwearied patience in eliciting the facts and testing the accuracy of the patient's statements; to Mr. Fothergill for the admirable drawings, which illustrate the case; and to Mr. Hudson for the excellent photograph.

## Commentary

*Is the patient malingering?*—The first question to be considered is, of course, whether the patient's statements are true or false. There is something in his manner which is little fitted to inspire confidence, and he has been often in hospital with various nervous symptoms, and I have been told that after one of his former visits he had taken some credit to himself for having outwitted the officials. Such considerations as these have led us to watch him very closely, to pay him surprise visits, to see him when he was not aware of being observed, to have an eye kept upon him by quite a number of good observers; and never once during the five months has any one of them seen him walk except in his three-step method, never once have they seen him awake without

his ankle clonus coming speedily into action, and only a degree of diminution occurs when his attention is absorbed as completely as we find it possible to do. I do not believe that any man could keep up, during a period of five months, clonus of the right foot, and irregular jerking spasms of the left and of the right hand, by a mere effort of malingering, and I think that a malingerer could not always remember to take the triple step, as our patient always does; and therefore I think his motor symptoms are genuine, and give proof of profound disturbance of the nervous system. The sensory symptoms were only discovered when his case was being taken, and all who have had to do with him have come to the conclusion that he is describing truly what he feels; and some of us have had a good deal of experience of judging of men, and are not supposed to be over-credulous. But while one cannot be absolutely certain as to the sensory phenomena, the vasomotor changes have a definiteness that cannot be overlooked, and could not possibly be simulated.

*Faulty localisation of sensory impressions.—Allachæsthesia.*—It is not necessary to repeat the description already given as to the misplacement of sensory impressions, but it will be remembered that on the left half of the body, and on the left arm and leg, skin impressions of all kinds, while acutely and accurately perceived, are referred to a different position from the real point of contact, and always to the same position,—in the case of the limbs, to the opposite aspect at about the same level. I have carefully searched for recorded cases of a similar kind, and have found none. I have inquired of many neurologists, but none have met with a similar case, and I have demonstrated the facts in the Medico-Chirurgical Society, in the clinical class, and in my wards

to many members of the profession, none of whom have had any similar experience.

The nearest approach to it is supplied by some observations of Mr. Horsley, in connection with lesions in the Rolandic area. He had found that in connection with disease of particular portions of that area, say, that for movement of the thumb, the patient not only exhibited the motor phenomena, now so well recognised, but also a certain deficiency of tactile sensibility in the skin over the paralysed part, and a tendency to refer impressions to a higher part of the limb than that actually touched. Thus, if such a patient were touched on the thumb, he sometimes felt it as if it were on the wrist.

The condition which approaches it most nearly is that which Obersteiner, of Vienna, was the first to describe fully, and to which he gave the name allochiria (ἄλλος χείρ). Our case obviously is not referable to that category; for whereas, in allochiria, a touch is felt as if it were applied at a corresponding point on the opposite side of the body, in ours it is on the same side, on the same limb, but on the opposite side of it, although at the same level. Allochiria has been studied not only by Obersteiner, but by Ferrier, Fischer, Hammond, Brown-Séquard, Leyden, Féré, Gellé, Gay of Bournemouth, and others, but perhaps most fully by M. Weiss of Prague. These observers have ascertained that allochiria may affect one kind of sensory impression or all varieties, that not only may touch, but temperature-perception, pain, tickling, be referred to the opposite side, but also that sight and hearing may be so transferred; and that not only may the skin, but mucous surfaces be affected as well, and that even reflex movements and electrical stimulation applied on one side may produce their effects exclusively or in part on the other.

The kind of explanation which has commended itself to most writers on allochiria is suggested by the fact that it is so often found associated with locomotor ataxy or other organic disease of the cord. It consists in the conception that, one or more of the paths for sensory impressions being blocked, a transference is effected by commissural fibres to the corresponding conducting structures in the opposite column, so the sensorium receives an erroneous impression. I doubt whether this explanation proves very satisfying to any one, but certainly it cannot apply in the case of our patient, seeing that the impressions are not referred to the opposite side of the body, but only to the opposite side of the limb.

It might be suggested that we should account for faulty localisation of sensory impressions by ascribing it to morbid action of the nerve endings or of the nerves. But if this explanation were correct, one would expect it to be associated with some alteration of perception in other respects, some loss of acuteness; but here no such abnormality exists,—the acuteness of perception is as good on the one side of the body as the other, it is only the localisation that is disturbed. It might be conceived that some faulty conduction in the spinal cord or in the higher sensory tracts might account for it; but if the true seat of morbid action were there, one would also expect altered sensibility of other kinds. It is obviously most reasonable to refer it to the sensory centres and to a disturbance of a sensory faculty, which is developed somewhat later than others. From the time of birth a child perceives painful and other impressions, but only by degrees does it learn to localise them. This faculty alone is interfered with in our patient. Now it is conceivable that a change might arise in the nerve cells which receive the impressions, or in the mysterious

borderland where the mental and the physical meet, but I should think that few would feel warranted in dogmatising as to which of these may be at fault in such a case as the present. But I would venture to suggest that this case may perhaps lend some support to the opinion that in allochiria, as well as in the condition under consideration, the fault is not so much in the conducting fibres as in a morbid action of the centres.

As to whether it is likely that any gross lesion exists in the sensory centres, one is scarcely entitled to speak, but I think that this and all our patient's other symptoms are functional, and I hope to see them disappear.

The name allochiria has proved so useful in relation to the symptom which Obersteiner described, that I have thought it well to try to coin an expression which may serve as a name for this kind of faulty localisation, and, after consulting with my colleague, Professor Butcher, have decided to suggest the name "allachæsthesia," from *ἀλλαχῆ* or *ἀλλαχοῦ*, elsewhere, and *ἀισθησία*, perception, as the most satisfactory term that occurs to us.

*The eye symptoms.*—The eye symptoms of this patient seem to me well worthy of attention. All the time he has been under observation, both eyes have exhibited a measure of staringness suggestive of exophthalmos. Sometimes it has been so distinct as to be unmistakable. The Von Graefe symptom of delayed descent of the upper lid has appeared from time to time. It was first noticed on 7th October, and afterwards disappeared, but of late weeks it has been constant. There is no anæsthesia of the mucous membrane of the conjunctiva or of the cornea, and the palpebral and lachrymal reflexes are normal. The pupils are of natural size, and act normally. The field of vision is very markedly diminished, both in respect of form and colour. Instead of a normal 90° on

the temporal, and 60° on the nasal side, the perimeter shows only 20° and 10° respectively in the horizontal line; while, instead of a normal 50-60° upwards, and 60-70° downwards in the vertical line, we have less than 20° above, and about 18° below. There is not much difference between the right eye and the left, but the contraction is rather more marked in the left. The field is also found to be distinctly wider when the observations are taken from the periphery inwards, than it is when they are made from the centre outwards; but by both methods the fact of the greater concentric contraction in the left than in the right is elicited.

He exhibits distinctly the phenomena of monocular diplopia. When an object is held near one eye, the other being closed, he sees it as one, but when it is withdrawn to a distance of from 12 to 24 inches a second image appears, and he says that the second is smaller and more hazy than the other. At greater distances this continues. I have found that when I made him look for some time at an object, say a pencil, he described it differently at different periods. He saw it first as one, and then as two, as the condition of the muscle of accommodation varied.

His perception of light seems quite good within the limits determined by his concentric contraction. His perception of colour is remarkably altered. The blue field is smaller than the red, and even than the green, but all of them are markedly smaller than normal. The only further peculiarity is that, while the field for green is smaller in the left than in the right, those for blue and red are slightly larger.

The various media appear to be normal, and the fundus shows to the ophthalmoscope a degree of pallor of the disc.

The condition thus conforms in a considerable degree

to what occurs in the group of functional diseases commonly spoken of as "hysterical." It is true that there is no blepharospasm and no squint, but these, although occurring in hysterical subjects, are by no means frequent. Again, there is in our patient no want of sensibility of conjunctiva, or of cornea, and that is rather a common seat of hysterical anæsthesia. Gilles de la Tourette cites a curious fact which goes to prove its frequency. He tells that some writers in the Middle Ages had ascertained that when the devil puts his mark upon a sorcerer, be it a witch or a wizard, that mark is often the depriving of certain parts of the body of sensibility to pain, so that needles could be put into these parts without causing uneasiness. And not only was it found that he often stamped these sorcerers on hidden and unclean parts, but he sometimes chose the noblest parts of the body, as for example the eye. Now it is clear that many of the unfortunates who were regarded, and even regarded themselves, as being colleagues of the devil, were really suffering from severe forms of hysteria, and the interpretation of their conjunctival and corneal anæsthesia accorded with the superstition and the want of medical knowledge of their times. It is surprising how complete the anæsthesia is in many of these hysterical subjects. Touching these very sensitive parts with the point of a pencil or a roll of paper, no pain is felt, and very commonly there is no reflex closure of the lid, or perhaps even no reflex lachrymal secretion.

But the concentric contraction of the field of vision is an important and common symptom. Dana says that retinal want of perception is the most frequent of all the hysterical anæsthesiæ. It is true that on the one hand such concentric contraction does sometimes result from organic disease, but in the majority of cases concentric contraction, or marginal scotoma, as it is often called, is

hysterical. Among the organic conditions with which it is associated, retinitis pigmentosa deserves the first place, but that is of course connected with characteristic ophthalmoscopic changes, and with dimness of sight, whenever the light is dim (nyctalopia), and neither of these conditions is present in our case. It is scarcely necessary to notice other organic local changes affecting the periphery of the fundus which might induce a somewhat similar condition, for the fundus is natural. Next in frequency comes atrophy of the optic nerve, whether simple or inflammatory in origin. But a marginal scotoma would necessarily, if so extensive as in our patient, be associated with considerable impairment of central vision, while here it is normal. Our patient's discs, too, show no changes corresponding to optic atrophy. Marginal scotoma might also be due to glaucoma, of which there is no sign in our patient. These organic conditions being thus set aside, we have to accept the view that the symptom is functional.

The monocular diplopia is another feature well recognised as characteristic of hysterical disease. The varying consistence of different parts or layers of the lens is said to account for the tendency for two images to reach the retina, and it is believed that the action of the muscle of accommodation is able, under certain circumstances, to correct this tendency, but often loses the power from slight paresis. If this be true, it seems also conceivable that conditions of spasm may in their turn similarly alter the lens, and induce diplopia from spasm. This is rendered the more probable from the fact that squint in hysteria is more frequently due to excess, rather than diminution of muscular action, and that paralytic is rarer than spasmodic closure of the lids.

The disturbance of colour perception is one of the best known features of hysteria. Normally, the field for

blue is the largest; next come in order yellow, orange, red, green, and violet. The hysterical changes are not constant, but among those most frequently observed are the very peculiarities shown by our patient, viz. diminution of the field for blue as compared with that for red. Not unfrequently it is found that violet is lost, and green almost or entirely so, while blue, yellow, orange, and red, although contracted, retain their normal position. Sometimes, although rarely, there is hemiachromatopsia, one-half of the field only suffering change, and sometimes there is complete achromatopsia, so that all objects appear as if of different shades of grey or sepia. Where dischromatopsia occurs in association with organic disease, as in locomotor ataxia, perception of red or green is usually the first to go.

Summing up, then, the results of our study of the eye in this and in other cases, we conclude that Field's condition is characteristic of hysteria.

*The causeless or spontaneous ankle clonus and other tremors.*—From the end of September 1893 up to the date of present report (17th February 1894),[1] the patient has suffered from tremors. The chief seat of this symptom is in the right foot and leg, which constantly exhibit movements corresponding to those of ankle clonus. The movements are always present when he is awake, and are produced by the alternate action of flexors and extensors of the foot.

All the deeper muscles appear to take part in the movement; the soleus and gastrocnemius do not come into play, while the tibialis anticus and posticus, the peronei, the long flexors, the extensor longus hallucis, the extensor digitorum, are all found to contract firmly. When the foot is held so as to prevent the ordinary

[1] The condition persists, 26th May 1894.

clonus movement (and this requires very considerable force), other movements proper to the thigh and hip set in, and a kind of rotation of the foot is produced by the sartorius, quadriceps extensor, gluteus maximus, and tensor fasciæ femoris.

The clonus movements are distinctly rhythmical, about seven or eight to the second, as is shown in tracing, Fig. 10, taken on 4th November 1893. It will be observed that the strokes are for the most part pretty equal, and that there is remarkable constancy in their rhythm.

Attitude has no effect, the tremor being as persistent when the patient is lying in bed as it is when he is sitting or standing. Sleep arrests the movement completely, but whenever the patient awakes, certainly within a minute or two of his awakening, it is fully re-established. We have only once put him under chloroform, and on that occasion the tremors ceased before he became anæsthetised, and when consciousness returned he had already complained of feeling cold before they recurred, but within three minutes of the administration being stopped they were in full play. We have sometimes seen them less intense, although never entirely absent, when we succeeded in interesting him greatly in conversation or otherwise, as for example when we were taking a sphygmographic tracing, or in determining the area of allachæsthesia on the trunk.

The left foot is also affected with tremor, but less severely and with a less definite rhythm. In November, the movements of that part were more like those proper to chorea, but now the tremor is more constant. In Fig. 11 are shown tracings, in which the irregularity of rhythm is very apparent, the rate being about $5\frac{1}{2}$ per second. In the early part of the illness the arms also, and especially the right, showed some choreic tendency, now it frequently shows a rhythmical tremor Tracings

of the movements of the right and left hand show seven per second in the case of the right, and a similar number, though less pronounced and less regular, in the left.

I have heard it broadly hinted that these tremors were regarded by some as voluntary. I am fully satisfied that such an opinion is fallacious. My reasons for arriving at this conclusion are, that he has been closely watched under all sorts of circumstances, by the resident, the nurses, and the clerks, who have taken the chief charge of his case, and they have never found the tremor absent. I cannot believe that any one could always, not in public only, but when alone, when lying in bed resting or reading, when in conversation, when occupying a quiet back seat in the chapel, when enjoying the Christmas entertainments, never once forget to keep up the movements. Nay more, I believe it is impossible for any one by voluntary effort to keep up a movement such as exists in the right foot for an hour, much less for all the waking hours of many months; and even if one could imagine this possible, one would have to conclude that this patient was able to keep up other jerkings of a different kind and of a different rhythm with equal constancy in other limbs. It does not, however, follow that our patient may not regard his tremors with a certain complacency, as neurotic patients so often do, still it seems to me impossible that any physician of sound judgment should regard the whole tremor as the trick of an impostor.

To what, then, is the tremor to be ascribed? It cannot be referred to mere action of muscles, nerve endings, or nerves. It must result from abnormal discharges of nerve force from motor nerve cells. But what cells,—those of cord, basal ganglia, or Rolandic area? It must certainly be admitted as possible that undue irritability of the motor cells of the cord might

account for such tremors as this patient presents, but, considering that all the other functions of the cord are normally performed, it seems improbable that it should be the real seat of the morbid action. We do not know enough of the relationships of the cells of the basal ganglia to entitle us to refer the tremors to them, but with regard to the motor cells of the Rolandic area the case is otherwise, and it is easy to conceive that an abnormality in them might induce the symptom. It is true that now it has come to involve both sides of the body, the features are less characteristic than they once were, but even now the prominence of the tremors of the right foot justifies the impression the features of the case first conveyed.

But if we venture to assume that the irritation starts from the Rolandic area, what may we suppose the nature of the morbid action to be? Is it organic, or is it functional? Even apart from the abnormalities of the eye and skin, one would at once conclude that it is functional, and, considering these associated conditions, no physician is likely to have any doubt on the matter.

During the past six years the relations of tremor to hysteria have been closely studied, especially in France. For, although there have been in the older literature occasional references to tremor in this connection, it is only since the rise of the Salpêtrière School under Charcot, and indeed in the later years of Charcot's working, that much has been written about it. In 1887, Dr. Ormerod exhibited in one of the London Societies a hysterical woman of 29, who, among other symptoms, showed a tremor of the hands closely resembling that of paralysis agitans. Hughlings Jackson concurred in the opinion that it was a case of hysterical tremor, and referred to the frequency with which the two conditions are associated. Letulle, Germain Sée, Axenfeldt,

Huchard, and others refer to individual cases; but Charcot[1] gave special prominence to hysterical tremor, and Rendu,[2] Pitres,[3] and Dutil[4] (one of the residents under Professor Charcot) published papers of much importance.

Pitres recognises three groups—first, hysteric tremors of the form "*trepidatoire.*" These, he says, are habitually localised in a limb, and especially in one leg. They are constituted by alternating, regularly rhythmic extension and flexion of the foot on the leg, the leg on the thigh, the thigh on the pelvis. They resemble the epileptoid trepidation of lateral sclerosis; they have the same amplitude, the same rapidity (5 to 7 strokes per second), the same uniform rhythm. They differ, however, in being unassociated with a tendency to spasmodic contracture. In some patients they present the peculiarity that they cease when the patient lies down, and occur only when he is sitting or standing, or *vice versâ.* They usually begin insidiously; once established, they persist for long, but may at any time be suddenly cured, or other changes, such as paralysis or contracture, come on. His second class is termed the "*vibratory*" form, and in it the tremor is constituted by very little strokes, short and uniform, giving the members movements of vibratory oscillation. They may occur anywhere in the body, but are most common in the arms. Sometimes one does not see them unless the patient is made to hold his arms extended in the horizontal position, then one sees in the fingers and hands distinct movements, which disappear when the arm hangs inert. They do not usually prevent

[1] *Leçons du Mardi a la Salpêtrière,* tome i. p. 398.

[2] "Notes sur le tremblements hysteriques et ses varietes," read before the Medical Society of Paris, April 1889.

[3] "Les tremblements hysteriques," *Progrès méd.* Paris, September 1889.

[4] "Contributions a l'étude clinique des tremblements hysteriques," in *Nouv. icon. de la Salpêtrière,* Paris, 1890-1891.

the execution of voluntary movements, but render them awkward and difficult, especially in regard to finer adjustments. They sometimes resemble the tremor of Graves' disease, sometimes that of Parkinson, sometimes that of old age, and sometimes that of alcoholism. His third class is the "*purposive or intentional*" tremor,—those which only occur when voluntary movements are attempted, and cease when the muscle is at rest, just as one sees in multiple sclerosis. Each of these groups he illustrates in his admirable paper by a variety of examples, and it will be seen that our patient corresponds to the first and second of his category, not to the third.

Dutil has also classified the tremors into three groups :—

| | | |
|---|---|---|
| 1. Vibratory Tremor—8-9 and more oscillations per second. | Persisting during rest; slightly, if at all, under control of the will. | Resembles the tremor of Graves' disease, alcoholism, and paralysis. |
| | *a.* Remitting purposive tremor, present or absent during rest, increased by voluntary movement. | Resembles closely mercurial tremor, and less closely the tremor of multiple sclerosis. |
| 2. Tremor of Medium Rhythm—5½-7½ oscillations per second. | *b.* A paraplegic form confined to the legs. | Resembles the spinal epilepsy, foot clonus of spastic paraplegia. |
| | *c.* A pure purposive tremor, absent during rest, occurring only with voluntary movement. | Exactly corresponds to the tremor of multiple sclerosis. |
| 3. Slow Tremor—4-5½ oscillations per second. | Persisting during rest; slightly, if at all, under voluntary control. | Resembles tremor of paralysis agitans and senile tremor. |

Charcot, writing after the publication of Dutil's paper, suggests a classification, which, he says, differs in no essential point from that of his assistant. It is as follows :—

HYSTERICAL TREMORS

| | | |
|---|---|---|
| *a.* Tremor not exaggerated by voluntary movement. | 1. Oscillatory Tremor—slow, 3-6 per second. | Resembles paralysis agitans or senile trembling. |

| | | |
|---|---|---|
| | 2. VIBRATORY TREMOR—rapid, 8-9 or more per second. | Resembles Graves' disease, alcoholic tremor, that of general paralysis. |
| *b.* Tremor, present or absent during rest, induced or exaggerated by purposive movements, which do not accelerate but augment the amplitude of the vibrations. | 3. PURPOSIVE TREMOR—intermediate as to number of vibrations as to No. 1 and No. 2. | Resembles tremor of multiple sclerosis. Resembles closely mercurial tremor, which Letulle believes to be very often a hysterical tremor. |

It will be observed that our case scarcely finds a place as readily under the classification of Charcot as under that of Dutil. But the cases present such variety that classification is extremely difficult. The really important point is the final establishment of the fact that hysteria is capable of reproducing all the varieties of tremor with which we are familiar in other nervous diseases.

The classification which seems to me the most convenient and satisfactory applies both to the tremors of organic and functional disease. It is the following:—

1. Rhythmical coarse tremors, occurring apart from voluntary movement, and to some extent under control of the will, such as are seen in paralysis agitans, and sometimes in senile conditions.

2. Rhythmical coarse tremors or movements occurring apart from voluntary movement, and little, if at all, under the control of the will, as seen in some functional spasmodic states.

3. Non-rhythmical coarse tremors, attendant on voluntary movement, and not under control of the will (volitional or action tremors), as seen in multiple sclerosis.

4. Non-rhythmical coarse tremors or movements occurring apart from voluntary effort, and not under the control of the will, as seen in chorea.

5. Non-rhythmical or convulsive movements or

tremors, commonly superadded to persistent rigidity of muscles, as seen in some cases of birth palsy and athetosis.

6. Rhythmical or non-rhythmical fine tremor, independent of voluntary movement, and not under control of the will, as seen in senile conditions, in general paralysis of the insane, alcoholism, mercurial, and sometimes in lead poisoning.

The tremor of our patient is referable to the second of these groups.

*The gait.*—The gait in the case of F. presents peculiarities which I have never seen in any other patient. It is perfectly regular in its rhythm, and quite simple in its character, betraying no unsteadiness, but a constantly recurring irregularity. Fig. 13 shows clearly the peculiarity. The figure was obtained by letting the patient walk, his stocking soles well saturated with whiting and water, the impression being afterwards photographed, when the whiting had dried in. I find this a very convenient method of recording certain of the facts of a gait, and am able to demonstrate by means of it such peculiarities as belong to hemiplegia with descending degeneration, locomotor ataxia, and cerebellar disease. It will be noticed that our patient zigzags, and that zigzagging is effected by the leg in movement being at each third step adducted, so that the foot which is being moved crosses in front of that on which the patient is resting, and is planted on the opposite side of the resting foot to that which it would naturally occupy. But then, in addition, it is to be observed that the step immediately following this adducted one is made more in the same direction as the adduction had brought it, because, the centre of gravity being made to fall within the area of the foot so abnormally placed, the body

swings to the corresponding side, and the other foot is planted further out. The next step rectifies this, and the foot, now advanced, is planted pretty much in the middle line along which the patient is intending to go. But then comes the adduction step, and this time in the foot which on the previous occasion of adduction had been the foot at rest, and this foot passes in front of the other, and, although carried sufficiently forward, is planted on the wrong side. Hence, again, the sway occurs away from the middle line, and so on through a series of steps, however long. He has some difficulty in turning round, but not so much as one often sees in cases of ataxia or of cerebellar disease, and the rhythmical march is always followed, except when he is nearing a chair, or his bed, or a door, when one sometimes sees him throw his arms out, and catch at the bed or table or other stable structure, and pull himself to his desired haven. This gait has persisted now for five months; it appeared quite suddenly at about the same time as the clonus, and he has never been seen to walk in any other way except under the circumstances just described. We have questioned him closely as to the reason of this peculiar gait, and at first he always said that it was due, not to any feeling with regard to his equilibrium, but simply because the tendency to the muscular movement was irresistible. Latterly he has asserted that he does it because he feels that he would fall over if he did not use the leg in the peculiar way. And yet it seems extraordinary that such a change of sensation should occur only with every third step, and that alternately in one direction and in the opposite. We shall return to the explanation a little later on.

In considering gait, it seems to be necessary to satisfy ourselves, first, as to the patient's mode of standing, his equilibrium; and second, as to his mode of walking

forwards and backwards, and to his mode of turning round.

In regard both to standing and walking, it is desirable to know how it is modified, if at all, by the patient having his eyes open or shut, by his having a light cane to guide, or a stick to support him, and by his holding in one or both hands a considerable weight. Sometimes the condition of the eyes makes a great difference; sometimes the patient is steadied when he holds a light flexible cane incapable of giving support; and sometimes the carrying of weight improves or diminishes the accuracy and vigour of the movements.

In describing the features of gait, it is well to distinguish the state of the foot, the legs, the trunk and head, and the arms, and this both in respect of walking and of standing.

Peculiarities of gait may be found due to conditions of the bones, conditions of the joints, conditions proper to the muscles, and conditions originating in the nervous system.

Physicians rarely have to do with abnormalities originating in the bones or in the joints, but have frequently to deal with those proper to the muscles, and still more frequently with those originating in the nervous system. I shall not attempt, in the present paper, to discuss any of the gaits originating in morbid states of bones, joints or muscles, but shall confine myself to the fourth category, *i.e.* those connected with morbid states of the nervous system.

These morbid states may affect (*a*) the functions of the afferent structures, (*b*) those of the nerve centres, and (*c*) the efferent structures, motor or co-ordinating. For efficient equilibration and progression, all these functions must be normally performed. We may learn a good deal regarding them by watching their development in

the child. It learns first to stand, when it is leaning upon the knee of the mother or nurse, and its early efforts are frequently interrupted by sudden yieldings of the knee joints, or by liftings of the foot, like the pawing of a horse. Gradually it learns to stand steadily, and, after a time, without support from the hands. But before it learns so to stand it has made some progress in walking. Holding on by some one, or by a chair or sofa, it begins to be able to put one foot before the other, and after a time, fixing its eyes on some desired object, it makes a little run towards it; and then gradually it learns to walk alone.

During this process it has been acquiring the power of appreciating various kinds of afferent impressions, and to use these as a guide in its movement. Certain centres have been educated to receive impressions and to emit impulses, and co-ordination of the various groups of muscles is laboriously acquired.

When disease of the nervous system interferes with equilibration and walking, it may be by failure in any of these functions.

1. It is often due to failure on the part of afferent nerves. If there be plantar anæsthesia, from disease of nerve endings or of nerves, one important guide is lost.

In cases of peripheral neuritis a like difficulty arises, although it is, of course, generally complicated by the occurrence of motor nerve change along with the sensory. Impairment of muscular sense still more markedly affects this function, because, although the impressions it conveys can scarcely be said to reach the consciousness, their reaching certain centres is essential to equilibration and walking. When either of the kinds of impression just referred to is at fault, the patient comes to guide himself largely by the eye, and when it is closed, equilibration threatens to fail, and walking becomes almost

impossible. Articular impressions, and impressions derived from the semicircular canals, no doubt also play an important part, and when they are in any way interfered with, equilibration and gait are disturbed. It would seem as if excess or perversion of impression in the semicircular canals more frequently disturbed these functions than does a diminution, as is testified by the phenomena of Menière's disease, with which the profession is now so familiar.

Few conditions, however, disturb equilibration and walking more than contradictoriness of sensory impressions. If the information conveyed by the two eyes differs, as in recently developed squinting, or if the eyes transmit to the centres an impression contradictory to that which is sent by the semicircular canals, or from the soles, or from the muscles, or from the joints, the sensation of giddiness necessarily arises, and equilibration and walking become impaired.

2. The fault may lie in the nerve centres of the cord, or of the brain, and the fault may be situated on the sensory side, or on the motor side, or on both. In cases of chorea, of cerebral or of cerebellar tumour, in other diseases of the Rolandic area, and in trade spasm, such as Treadler's cramp, we find illustrations of the conditions.

3. The fault may be situated in the efferent structures, motor or co-ordinating, as we occasionally see in cases of locomotor ataxia, where sensation remains unaffected, and often seen in spastic paralysis, where the lateral columns are sclerosed.

We are now prepared to return to an analysis of the features of our case. The patient, in standing, equilibrates quite well, even with the feet together, and would be practically steady, but for the clonus. With the eyes closed some degree of swaying occurs. The taxo-

graphic tracings (Fig. 12) show the distinct increase of clonus, and the degree of swaying that probably attends upon that increase which follows the closing of the eyes. The legs in standing show no change except the jerking, and this is also true of the trunk and head. The arms exhibit their own tremor, and are held a little out from the body in a somewhat unnatural way. In walking, the peculiar rhythmical alteration already described manifests itself. The planting of the feet always follows the same rules, the toe of the adducted foot being turned more inwards than it ought to be in normal walking. The legs show no peculiarity, except the abnormal adduction and its consequences. The same is true of the trunk and head; but the arms are held a little out from the body, much more than in standing, and in a way that a person does who is not sure of his equilibrium. The closing of the eyes gives a degree of uncertainty to the gait, but does not alter it more than one would expect in the circumstances. He derives no help from the use of cane or stick; and when two students support him, and try to keep him steady by grasping his arms, his movements become more tumultuous, and more like those which one sees in chorea major. The effect of carrying weights is not important, making little difference, if any, in his walking.

Such being the facts, how are they to be explained? We cannot refer them to an abnormality of the afferent nerves, because these nerves, excepting in respect of the allachæsthesia in part of one side, are normal; neither can we refer them to a fault in the efferent nerves, for a mere nerve change could not explain them. They must be explained by a central change. That might be sensory, motor, or co-ordinating. Now the patient has no giddiness nor other sensation which could induce the movement from side to side, and therefore I incline to

acquit the sensory function. On the other hand, he performs with each third step a peculiar superadded movement of adduction, which necessarily disturbs his equilibrium, and would make him fall over, if he were not sustained by the other leg, which is rapidly thrown out and firmly planted. Thus it seems to me that the peculiarity of gait, like the clonus, results from a rhythmical discharge from motor cells, occurring periodically and on the two sides alternately. It may be that such a discharge proceeds alternately from the two Rolandic areas, but it is also possible that the motor cells in the cord may be at fault.

I have searched medical literature, and particularly works dealing specially with gait, such as those of Vierordt,[1] Gilles de la Tourette,[2] and Paul Blocq,[3] and have found no record of an analogous condition.

*The vascular changes.*—The symptoms proper to the vasomotor function are among the most interesting. They have not continued equally distinct throughout the months the patient has been under observation. It is true that all the time factitious urticaria has existed, but the variation in the size of the thyroid gland was much more distinct in the earlier weeks of the illness than of late. It is interesting to note that the factitious was unattended by any trace of ordinary urticaria. The explanation of the vascular phenomena evidently lay in an occasional abnormal dilatation of vessels, manifesting itself in the skin generally when the surface was rubbed, and in the thyroid under conditions of emotional excitement. Along with these changes one naturally associates

[1] Hermann Vierordt, "Das Gehen des Menschen in Gesunden und kranken Zuständen."

[2] Gilles de la Tourette, "Etudes cliniques et physiologiques sur la marche."

[3] Paul Blocq, "Les troubles de la marche dans les maladies nerveuses."

the prominence of the eyeballs and von Graefe's symptom of delayed descent of the lid, which in the end became so prominent.

*The nature of the process.*—Careful consideration of all the facts of this case leads me to believe that it is functional rather than organic, and yet it appears quite possible that organic changes may be in process of development. There is no doubt that the general health is worse than it was when he came under my care. He has lost weight, he is paler, his expression has become more anxious; indeed, his appearance produces quite a different impression on the mind from what it did at first, more favourable as to his character, less favourable as to his prospects of recovery. It is clear that disturbance of so many functions implies morbid action in many parts of the central nervous system, and if so many parts were diseased in consequence of organic change, the life could scarcely have been prolonged as it has been. Again, if the symptoms were due to organic change, they could not have remained so constantly the same as they have done, but during the months they must have shown greater alterations. On the whole, I regard the case as a fine example of Hysteria in the Male.

*Prognosis.*—My hope has been that we should see improvement set in ere now, but as yet there is no sign.

*Treatment.*—The treatment appears to have entirely failed, unless the improvement in the thyroid was due to it. I first sought to produce a strong impression on the patient's mind as to the usefulness of setons in the back of the neck, and then introduced a seton in that situation, but beyond causing great inconvenience and much discomfort to the patient, and possibly affecting the

thyroid favourably, no result followed. I have thought it right to try the effect of hypnotism, and, with the aid of various friends, have tried to bring him under its influence, but we have not succeeded as yet in producing the full effects; a measure of improvement has occurred after one or two of the séances, but only of a temporary character.

THE END

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